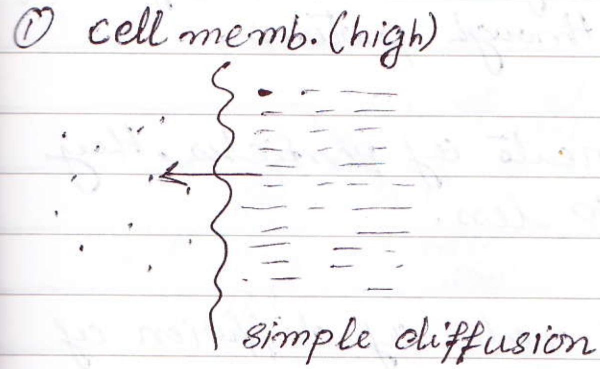
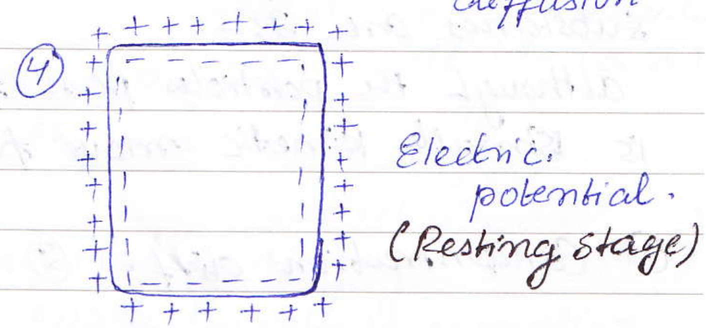
"Membrane physiology & Muscle"

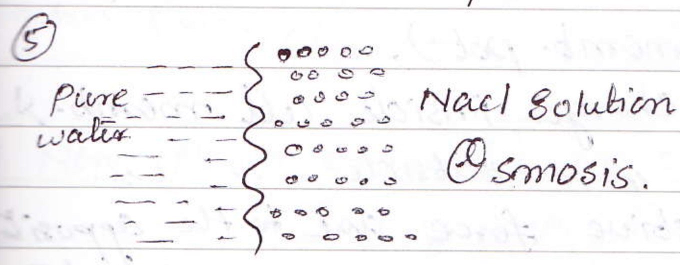
16/9/013
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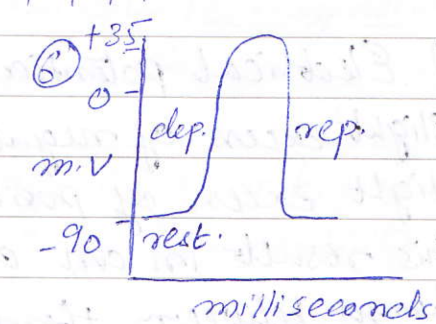


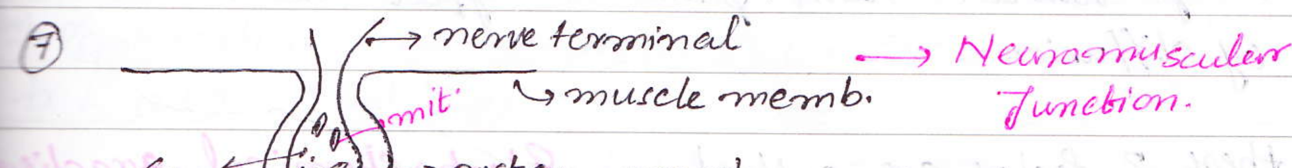


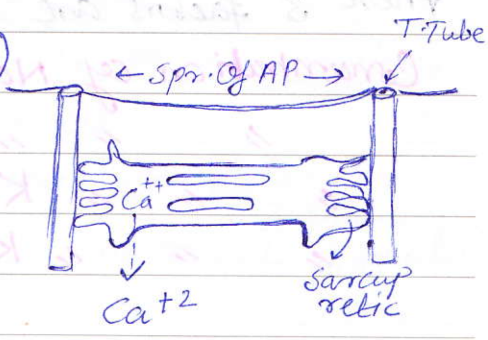


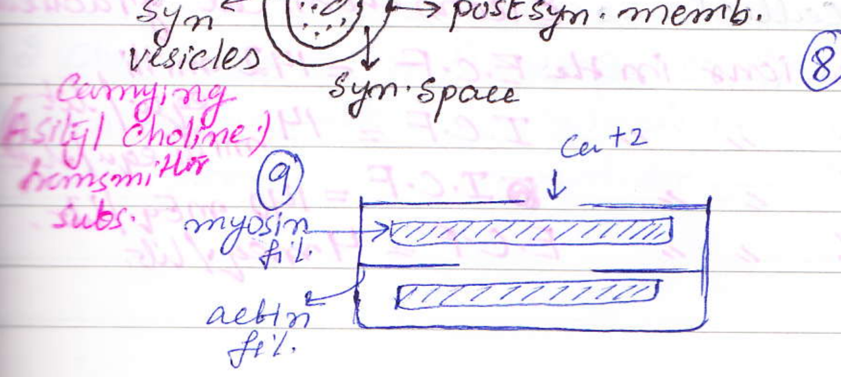












lipid substances can pass through memb. easily through lipids, while water soluble through proteins.

By kinetic, molecular movements of particles, they can pass from higher conce. to less.

(4) Factors that affect the net rate of diffusion of substances are 3:-
although the particles pass from high to law but there is small kinetic mov. from law to high.

(1) Concentration diff. (2) pressure diff. (from higher pressure to lower p)

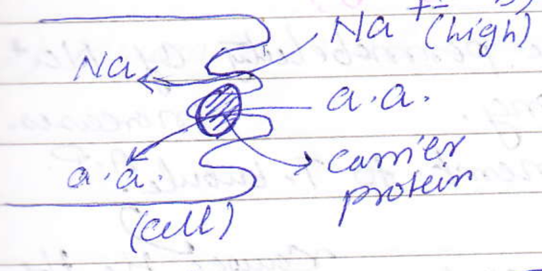
(3) Electrical potential (memb. pot).
(slight excess of negative charges inside cell memb. & slight excess of positive " outside " ".
this result in an attractive force due to the opposite charges between them. & this can effects the net rate of diffusion.

these 3 factors are called → Electrochemical gradient.

Concentration of Na ions in the E.C.F = 142 milli equ/lit.
" " " " " I.C.F = 14 milli equ/lit.
" " " K " " I.C.F = 140 mEq./lit.
" " " K " " E.C.F = 4 meq./lit.

"Active Transport":- 1) Energy ATP (Break down of ATP.)
2) Carrier proteins.

(primary A.T.)



b) Sec. A.T:- Na+ ions outside conc is high so ions will transp. according to conc. gradi. but carrier p. will transport another molecule with it. (a.a) called:- (Cotransport)

another type of Sec. A.T) Same as previous but carrier proteins will transfer the another subs. outside the cell → called (Counter transport).

"Receptor (Resting) memb. potential":- of large nerve fibers. = -90 mV. (means inside the cell the negative charges is more than positive charges).

Nerve (Action potential:) rapid change in memb. potential.
1) Resting m.p. 2) depol. stage. (permeability of mem. to Na+ ions increase (Influx).
3) Repolarization:- increase permeability to K outside (Outflux). → resting stage (-90mV).

(- + / - + Na+ / - + / - + / - +)

2 Charac. of A.P:- 1) propagation of A.P. (The cell memb. is known as Excitable cell), (muscle or nerve cell.)
These propagation is known as → Nerve Impulse or Signal. or muscle Signal.

for stimulation.

Threshold → الحد الذي لازم الوصول اليه لكي يحدث A.P. وهو مثلاً 65- لازم نزود الجهد من 90- الى 65- لكي يحدث A.P.

Asetylcholine increases the permeability of Na^+

Excitation - Contraction Coupling.

A.P transfers from muscle memb. to T. tubule. A.P. increases. causes the Sarcoplasmic reticulum to release Ca^{++} & rep.

actin & myosin slide among each other & contraction takes place, Ca^{++} return to it's place

Types of smooth muscle → 1) Multiunit. 2) Unitary S.M. (Visceral or sensitial smooth muscle).

Seperate muscle fiber & each one is innervated by single nerve ending & can contract independently. (Iris of Eye, Cilliary muscle of Eye, Pylo electric muscle. Erection of hair when stimulate by sympat.)

2) Visceral → mass of 100-1000 muscles fibers connected to each other by there cell membranes & they are joined by gap junction allows free movements of Ions, so that propagation takes place to the whole muscle as a single unit.

* Skeletal muscle fibers stimulated by only nerve signals.

Smooth muscle → by 1) by local tissue chemical factors, like decrease in O_2 or increase in CO_2.

2) Hormones. 3) nerve signals. 4) muscle stretch.

"Chapter (2)"

(*) "Cardio Vascular System" :-

18/9/013
Wed.

"The Heart as a pump"

~~during the~~

Isotonic , Isometric

(the tension is same but the length changes).

Isometric → same length.

(*) The Heart :- as a pump. The 2 atrium, the two ventricles

Septum muscular between "Atrium & Ventricular but between atrium & ventricular fibrous system, impulse can't travel through it but only through special nodes.

(*) Type of muscles in the heart :- (Cardiac muscles).

(Atrial muscle, Ventricular muscle) → (contraction same as the other muscles but here contraction is prolonged. comparing to skeletal muscle)

Conducting muscle (special). → non contractile but conducting only. Skeletal [illegible] muscle. because can't contract at all.

Cardiac muscle act as Syncitium all the network works together. The most Imp. thing in (Syncitium) the (Intercalated) disk + gap junct. between 2 adjacent cell membranes which makes gap junctions., diffusion of ions very fast from one cell to other cell very rapidly.

→ Why the atrium & Ventricular has diff. Synsitium.
due to the presence of fibrous tissue.

Difference between action potential between Skeletal & Cardiac muscle :-

Na, K channels, fast channels.

but in Cardiac muscle the contraction is prolonged due to the presence of (platue) (Short pause) (contraction at the end of depolarization).

In Cardiac muscles → Na channels fast but Opening of Slow Ca^{++} channels (Sodium-Calcium channels) also membrane permeability to K^+ decrease.

* platue :- 1) decrease of K^+ channels.
  2) presence of Slow Ca^{++} channels.

A.P = 0.2 - 0.3 msec.

* Refractory period → Time during which no restimulation can take place to muscle.

1) Absolute R.P. → we can't restimulate.

~~Refracto~~

2) Relative R.P → (restimulation & reexcitation can take place. If there is strong stimulation at the end of repolarization)

(Refractory period in atrial muscles is less than in ventricles. Every thing in atrial muscles are less than in ventricles.)

⊛ "Excitation contraction coupling":-

→ Action potential spread through muscle membrane

What are the difference between the E.C.C between Cardiac & Skeletal?

1) Source of Calcium. ⊕-

2) T-tubule in cardiac muscle are much larger than that found in skeletal also T-tubule in cardiac contains fluid which have polysaccharides. (-) charged which attaches to (+) calcium.

3) Cardiac muscle source of Ca E.C.F but skeletal muscle ~~contains~~ source of Ca T-tubule.
but the sarcoplasmic reticulum is not well developed although it's large (diade) not triade.

4) duration of contraction.

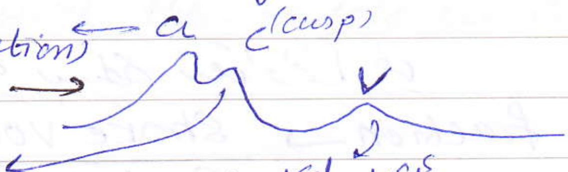
⊛ The Cardiac cycle → (mechanical events → contraction) from beg. of one heart beat to the beg. of sec. heart beat.
Atria is known as primer pump.
1) The first who contract 2) because it's the major
from it the blood distributes to the other parts of heart

(80 %)

Filling of Ventricles :- ① Rapid passive filling.
(due to ← ② Continious slow filling. (diastisis)
pressure gradient.) ③ Active filling (20%) (contraction of atrium)
Increase in the ventricles so the blood can't pass freely due to increase in B.P in ventricles.
the closure of valve takes place due to (passive closure due to presure.) ??

Intra fibrillation → the contraction is not accurate
the atria can't contract the last 20% of blood
during rest normal, but during Excercise not enough.

Jugular nerve meter →



قبل vent cont. الدم يرجع إلى الخلف   أثناء إنقباض الـvent
Back word flow of blood, cusp. & close of valve.

Ventricular Emptying :-
1) Isovolumic Contraction (systole) no charge in volume.
Increase of Tention in Ventricular muscle but the volume is same. the blood can't enter the Semilunar valve only, if the pressure is high. after the tension reaches maximum the semilunar valve opens & the blood reaches the aorta.
2) Rapid Emptying strong contraction, after that the all the blood transfers to the arteries, presure gradient

in arteries becomes more than in ventricles so blood returns back to ventricles & closes the Semilunar valves

Why the closure process in

End diastolic Volume :- (preload) → The load on heart before contraction
آخر كمية دم موجودة فيها ال
لحظة في Ventricle انتهاء ال diastole

* Stroke Volume.

The blood that remains in ventric. after ejection → End Systolic Volume ← اقل كمية متبقية في

Ejection fraction → $\frac{\text{Stroke volume.}}{\text{End diastolic Vol.}}$

Is it possible to have diff. valu. for the above para in a healthy heart?? Yes according to the condition during Excersize the values may change.

Semilunar valve → more Stronger than A.V valve (mitral) that's why doesn't require papillary muscle.

Mitral valve → very soft & weak due to that bacteria easily can accumulate there & cause Rheumatic.

papillary muscle & Chordae tendiae → Only controls the closure of valve

Purpose of
Valve → prevents the back flow of blood.
Heart Sound → due to closure of valves, vibration of fluid. beside it which spread into the chest.
The first heart sound slow → A-V valves.
" Sec. " " hard → Semilunar (Stronger)

pre-load → End diastolic pressure from End diastolic Vol. pre-load ∝ E. diastolic. p ∝ End dias. volume

After load → Resistance in circulation
The load against which the muscle its contractile force.) ??

Regulation of heart pumping:-
1) Intrinsic mechanism (Frank Starling) 2) Extrinsic mech.
((within physiological limit, heart pumps all the blood that returns to it by way of veins))
① → The heart output is related to the venous return.
② → The Output of heart is related to the amount of stretch of heart muscle, but within physiological limits, but after that damaging of cardiac muscle.

③ → Atrial pressure.

S.A node, A.V node
? cardiac muscle as whole.
(S.A node, A.V node)

Sympathetic, parasimpathetic.
. Stimulation of Sympathetic

Stroke volume → ① Frank limit. ② Stimulation of Symp.

Sympathetic

Ca^{++} increase → Increase contraction.

⊛ What makes the heart act as a syncitium (network) ??
1) The Intercalated disk which forms between 2 adjacent cell membranes.
2) The Gap Junctions.

⊛ Why does the atrium & the ventricle has diff. Syncitium?
→ due to the presence of fibrous tissue.

⊛ Why does the action potential in Cardiac muscle differ from that in Skeletal muscle?
1) due to the presence of Slow Calcium channels.
2) And decreased K^+ outflow.

⊛ Excitation-Contraction coupling differene between Cardiac muscle & skeletal muscle :-
① Source of Calcium :- In Cardiac muscle E.C.F, while in Skeletal muscle T-tubule.
② T-tubule System. 3) duration of Contraction.

Filling of Ventricles :- ① "Rapid passive filling" (80%)

When the ~~right~~ atria is filled with blood from all parts of the body & from the lungs the pressure inside it increases & the blood tends to flow rapidly & ~~due~~ due to the pressure gradient towards the ventricles after the opening of the valves due to the pressure.

② Continous slow filling (diastisis) :- When the blood fills the ventricals the pressure inside the ventrical, increase & the blood from atria can't pass freely due to starts to the change in the pressure gradient.

③ Active filling 20% → The atria contracts & Empty the remaing of the blood to the ventricals, due to the increase in pressure on ventricals the "passive closure of A.V valve takes place".

Emptying of the ventricals :- ① Iso volumic Contraction :- Because no emptying at this time, due to the closed semilunar valves require time to open. The volume of blood in the ventricals remains the same but the pressure (tension) changes. The pressure Increases in the ventrical which leads the blood to open the semilunar valves & the blood goes to aorta or to the pulmonary artery.

~~Strong~~ ② Rapid Emptying (Strong Contraction) :- for the remaing of the blood. but still a, small volume of blood remains in vent. Known as End Systolic Volume.

The pressure in the arteries Increases, & blood tends to reenter the ventrical but the semilunar valves is closed by high pressure.

⊛ Why does the regulation of the heart pumping is imp?

→ Because if the mechanism of heart pumping was the same in all conditions for eg. during Excersice extra exhaustion takes place & require more Oxygen Supply, so if the pumping mechanism was the same hypoxia would take place, thats why regulation is imp.

⊛ Sympathetic Stimulation:- When heart requires more blood & Oxygen Symp. N. system is Stimulated & the Cardiac output (Stroke) increase. Heart rate + contraction increases. Cause Sym. N.S is spread on S.A node, A.V node., and the whole muscle fibers.

⊛ parasympathetic Stim. :- Spread on S.A node & A.V node & only a few muscle fibers of heart, so it's stimulation decreases the heart rate mainly rather than the contraction. (doesn't work on normal condition).

⊛ Function of the platueae:- To prolonge the time of contraction in the cardiac muscle 15 times more than in skeletal muscle.

Chapter (3)

Rhythmical Excitation Of the Heart.

— x — x — x — x —

The Specialized excitable muscle function → generation & conduction of Impulses.

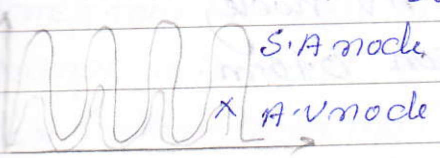
The Heart is able to generate its own impulse. (Autorhythmic Cells).

1) Atrial ~~[illegible]~~ contraction before ventricle.

2) Ventrical should contract simultaneously

Internodal fibers, S.A node, A.V. node, bundle of his, Purkinje fibers, The first part of ventricles which is conducted the Septum, then goes to the apex then to the base.

S.A node → Self excitation, the rate of discharge makes S.A node pacemaker, coz this contains the highest rate of discharge. 70 - 80/minute, A.V node do some self excitation but it cancels due to the presence of high rate of S.A node, A.V, & bundle in sepressed state



In Self Exciting cells the resting potential is less negative than the normal resting pot. −90. → −40. it's known as non resting membrane ✓ due to the presence of leakage of Na ions the negativity decrease & the potent reaches the Threshould without any triggering.

There is no rapid Na channels & the resting memb potension is also less neg. that's why there is no spike.

A.P doesn't reverse due to:-

1) Refractory period of cardiac muscle is long. so if the induction reverse the R.P isn't completed yet.

2) due to the presence of fibrous system.

muscles of heart are arranged in spiral form. so the transmition of A.P in angular form. 2 sheets of muscles separated by a fibrous septum.
conduction Impulses from apex to base.
Ectopic pacemaker → any structure other than S.A node that can act as pacemaker in abnormal conditions.
A.V node or any other structures other than S.A node if:-
1) S.A node is damaged. 2) If the rate of A.V node is higher than S.A node.

Sometimes the perkinje fibers (Ectopic focus) increase in rate 40mph S.A node remains 70 but ventricals contract very fast (Heart mermmar) no specific pumping.

If S.A node is blocked the A.V node will lead the conduction but in less effeciency the person will suffer from weakness.
the atrial contraction is not done only by sympathetic nerves. but this doesn't coz any defect coz 80% of blood passes to the ventricals directly

A.V node is blocked, S.A node works at rate of 70 & the Purkinje 30 conducts the ventricals, ventricals & Atria contract not simultanously, abnormal.

hyperpolarization → returns the memb. to resting potential due to opening of K^+ channels. which remain open for a long time that's why returning to the rest is not rapidly.

This occurs in all conducting nodes but rapid in S.A node

A.P from S.A node ~~to~~ of Right Atri to Left Atrium.
↓ direct transmition ↓ Inter atrial trans.

from Left Atrium to ventricals → Inter nodal bundles (3).

Conduction velocity depends on → 1) no. of gap junction
2) The diameter & the size of fiber.

A.V node → post. wall of R.A behind the opening of Tricuspid valve.

A.V node → Transitional bundle entering the fibrous skeleton.
↓ delay in conduction from S.A node to A.V node
total the delay due to less ~~of~~ gap junction 0.16m & small fibers.

Perkinje fibers → goes to the Endocardium & then only to 1/3 of myocardium.

The rate of conduction of perkinje → very rapid due to lots of gap junction & large muscle fibers & cause the ventricle are large so rapid conduction

. Refractory period of skeletal muscle is = to the contraction.

(many causes of these abnormalities)
medicines work on the generation & conduction of impulse

~~Conduct~~ Control of Induction by Nervous System.
Parasympathetic decrease the heart rate (Vagus nerve) release Acetyl choline increase the K channel opening Increase in the negativity due to the hyperpolarity, decrease the heart rate rapidly but decrease contraction ~~for~~ little, & when excessive stimulation of parasympathetic takes place heart rate stops but for a while but then purkinje fibers triggers
Sympathetic → norepinephrin opens Na channels increase in the A.P ↑ heart rate & contraction also increases.

—✗—✗—✗—✗—

Chapter (4)

[?]/2013
[?]ednesday.

Physiology of Cardiovascular system ("The ElectroCardiogram" (ECG / EKG))

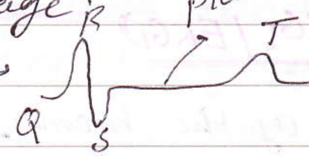
surface
- Recording of Electrical Activity of the heart. → ECG.
- squares containing paper → waves + Interval Signals.
- One Electrode area should be positive & the other negative
- so that the Electrical current flows from depolarized to (waves) (partialy re or partialy de) repolarized area.
- No current flow → when contraction is stable, either all depolarized or all repolarized.
- P wave, QRS, T-wave.
  - Atrial dep.
  - 3 depending on area of depolarization.
  - Q → septum, R → all except the base, S → base.
- but sometimes they aren't appear completely only R appears
- The depolarization of atrium don't appear coz it's predominantly under the QRS complex.
- P → Vent. repor.
- Diff in Voltage due to → 1) Vent. dep. high coz the muscle of the ventrical is thick long time. 2) Long repolarization or Putting the Electrodes directly on heart → high depolarization. A.P. directly.
- PR Interval → beg. of dep. of atriam, beg. of dep. vent.

- ST Interval → very imp in myocardial Infarction.
- R-R → complete cardiac cycle.
- wave → Voltage, Interval → time.
  - 0.12 - 0.2 → P-R Interval.
  - 0.16 → delaying of conduction.

QRS → 1.5 Voltage

plateu (no waves).

ST segment →



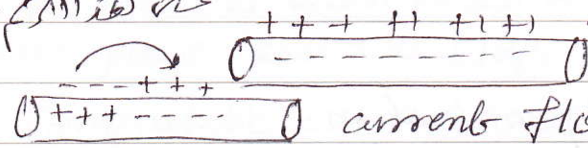
means ECG only records from de to re & not if both are equal

QT → beg. of Q till the end of T → de. & re of ventricl simult.
→ Imp. for heart rate.

← نحسب عدد المربعات الكبيرة وبعد ذلك نقسم 300 عليه.

$\frac{300}{4} \rightarrow 75$ (Big squares)

← عد المربعات الصغيرة ونضربها في الوقت 300 = 0.04 sec ——— نقسم 60 على هذا الرقم



→ Resting.

current flow,



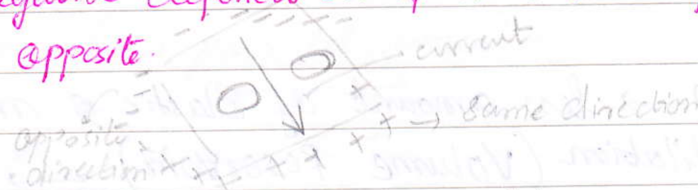
→ dep.

→ Constant no current.

flow of current from septum to the wall of ventricals in Elipsoid form.
septum → neg. ; wall → positive.
but after summation. result → from base to apex.
negative S wave
Conduction from Base to apex → positive if not negative
Repolarization in Ventrical muscle → from outersurface to inner thats why T-wave (positive).

⊕ The Electrocardigraphic leads :- Bipolar → 2 Electrodes (−)/(+)
One one right arm, other at left arm.
Unipolar → Positive Electrode on one arm & the other (0) potential to 3 other parts.
Augmented Unipolar → One on Right the other to left arm & leg.

4 Intercostal → lead one at right, to left ②
4,5,6 $\xrightarrow{aVL, aVF}$ 5th Intercostal space.
aVR → all the waves negative depends on if the current flows the same direction or the opposite.



The cardiac axis →
Vector → not always same, diff. from person to person but should be within limits. 59°, but may deviate to -20°.

① Standing or sitting position (Right or left) ② fat person → horizontal
③ Respiration. (pregnancy). thin person → vertical.
normal (Body size).

hypertrophy → Increase in size → abnormal.
Bundle branch block → shifting to the other side during blockage.

Overview of the Circulation, Medical physics of pressure, Flow & Resistance :-
30/9/013
Monday.

Benifits of close circuits → Any change which occurs at part of circuit reflects on the sec. part.
The same Quantity of Blood pumped returned back to the heart.
Control of Circulation → According to the activity of the Tissue & the need of the tissue.
For example during resting after eating the Blood will go to GIT, during excersize to muscles ... etc.

Arteries main characteristic →
strong Elastic vessel
(pressure Reservoirs).

distribution of blood under high pressure.
Artriol → bulk of musculer region, high innervation
high dilations & constrictions → control of Blood p.

Vein → less amount of Elastic & muscles → less constriction
& dilation (Volume Reservoir) → تستوعب كمية كبيرة من الدم.

(*) Cappillaries → pores.
Venules → between cappillaries & Vein.

Vein-Venules → The largest amount of blood in the Systemic circulation larger amount of Blood than Pulmonary circulation.
→ putting all the arteries & Veins adjecent to each other & calculating the total area of the (Cross section area).
Cross section area from the arteries till the Cappillaries decrease.
Velosity $\frac{1}{\alpha}$ Cross sectional Area.

Cappillaries → more bigger Cross sec. Area required for the Exchange process.
pressure in Pulmonary circ. less then Systemic:-
(1) The distance from lung to heart shorter.
(2) Enough pressure to expose to the Alveolar for exchange. while in cappillaries high amount of Pressure.

Pressure $\frac{1}{\alpha}$ Cross sec. Area.

→ increase Blood flow but not the pressure.

The diff. in pressure → (pressure gradient) causes the flow of Blood even if the values are diff.

Blood Flow → laminar → ((in parallel lines))
the layers ← parabolic profile
near the lumen
passes more rapidly than that near the wall due to the frictional factor. (resistence).

→ Turbulent flow ⇒ irregular (Sharp turn) 1) at Branches
2) In) Obstructions (wounds) على الشرايين (عكس الجرح، الغبار العقلي)
3) from large area to small area.
diff. in velocity.
(Normaly & Abnormaly inside the body)

B.P → distension of wall & flow of Blood forward.

Resistence → $\frac{\text{diff. in pres.}}{\text{Blood flow.}}$ → 2 values should be fixed & the 3rd variable.

Resistence → In pulmonary less than systemic due to the distence.

(Conductance ∝ $\frac{1}{\text{Resistence}}$)

Vessel diameter if increace → resistence decrease, conduct. Increase.

← إذا زاد القطر بمقدار ١ يزداد الـ flow بمقدار (1⁴).

2/3 of resistence in the Circulation found in Arteriols

1) primary (Essential) → due hyperpre. high ability to dilate & Constrict bcz
2) Secondary → due to age / due to diseases. very narrow resistence

Viscosity → depends on the, percentage of R.B.C. cells & proteins Increase Secondary.

Blood → highest Viscosity.
Viscosity Increases resistence & decrease Blood flow.
(due to increase friction)

polycethemia → Increase R.B.C, increase Viscosity hypertension.

Heamatocrit → ~~goes~~ Total amount of cells in Blood.
Abnormal & normal → 1) high Attitude.
2) pregnency.
3) Smoking.
4) Excessive (dehydration) → Concentration of R.B.C
Co2 plasma is lost as sweat

(Cancers)
(Leukimia)
very much increase in W.B.C

Alveoli, ﺗﺒﺎﺩﻝ ﺍﻟﻐﺎﺯﺍﺕ

But ~~due to~~ the Infect.
not very much increase in W.B.C.

Sympathetic → more in Artriole ~~St~~ (Constriction)

decrease stimulation or ~~increase~~ inhibition diam. → Increase diamets but no Parasympathetic Innervation.
→ more pressure because flow law.

"Vascular destensibility & Compliance"

2/10/013
Wednesday

→ fractional Increase in volume of the vessel
for each millimeter of mercury rise in pressure.

الفرق الناشئ بين الـ Volume القديم والزيادة نتيجة الزيادة للضغط.

$\frac{\text{Increase in Vol.}}{\text{Increase in pressure}} \times$ ~~Increase pressure~~ Original Vol.

~~The Original Vol~~

Accomodate the perstaile nature of B.V → Imp. of distesbility.

① during systol the out-flow of blood occurs but during diastol the B.V return to the original place which gives another pushing force for blood.

distesibility of Veins is larger than arteries due to thickness of wall.

Compliance (capasitance) → the amount of fluid (volume) that can be stored in the vessel as a whole.

Capasitance in Veins high due to → large volume.
Compliance = dis x Volume. → large distensibility.

There is strong relation between pressure & Volume in arteries :-
Small change in volume causes a large change in pressure.
In Veins → a large change in Vol. causes a small change in pressure.

Note:- In Vessels there's no parasympathetic sti.
Increase in Sym. stimulation → (Vasoconstriction, increase pressure, imp. in the cases of Heamorage, decrease in Vol. of blood cause decrease in pressure, so sym. stimulation incr. the pressure

during Excersise increase in vaso constriction.

* "Phenomena of Delayed Compliance & Reverse delayed
If there's any sudden increase اچانک اضافہ Compliance
or decrease in volume of the smooth muscle sudden
stretch occurs after that it has the characteristics of
reversing it's original length. eg. → 1) Blood Transfusion
delayed Complianced & Reverse of S.M all over the body. { → 2) during the desire to pass urine.

* Blood Transfusion → slowly to prevent the sudden increase
in pressure but here may → Reverse Delayed Compliance
haemorrage → Reverse delayed. can take place

Pulse pressure → diff. between systol & diastole = 120 − 80 = 40.
As we go further from the Arteries to cappillaries, Pulse pressure decrease due to decrease in pres.

* Factors affecting pulse pressure:—

1. Stroke Volume ∝ pulse pr.
2. Compliance $\frac{1}{\alpha}$ pulse pre.

eg:- 1) Arthrosclerosis → systolic pressure increase due to the increase Compliance due to deposition. ↗ Increase in systole.
2) Aortic Stenosis → Stroke volume decrease due to small diameter.
3) Patent ductus Arteriosus → Increase in diastole
4)

diastole & systole ليس عامة
الأولى
Mean → أخذ عدد من الأشياء وأخذ متوسطها — قياس الضغط عدة مرات ومن ثم أخذ المتوسط. نفس الشيء

MAP = 1/3 Pulse pressure + Diastolic Avereage = قياس مرة واحدة
being 60% from diastole pressure, & 40% from systole

CVP = (0) ⟷ Blood in Right Atrium.
Pressure in Veins → +4 pressure are found → (1) due to the compression of veins in the neck, abdomen by diff. organs, increase the resistence & thus increase pressure +4.
(1) Heamorage (2) Increase in Contractibity of R.V → increase the out put, R.V empties, Blood transfer from R.A to R.V due to p gradient R.A pressure decrease.
If Pr increases in R.A, it — decrease pressure in R-A

Hydrostatic pressure → due to gravity, at level of R-A, as we go down ward till abdomen + pressure, upper wards in (−). If the H.P between R.A = 0 till level of foot = 90.
If there is compression we add +4.
upward (−) pressure. at neck it's (0), due to compression,
Rule :- (−) pressure → suck's, (+) pressure → pushes.
Skull → no compression (−) pressure, thats' why during operat sinuses shouldn't be opened.
Mean pr → act on Arterial & Venous system

Venous Pump :- Increases even if slight mov. takes place coz the veins are located near the S. muscles which occurs back flow & due to valves pr

Causes of swelling. (1) standing still without mov
(2) pregnancy → Edema.
Increase in the Uterus size → increase in I. Abdominal Pressure
(Varicose Vein) ←
during Excersice → Blood goes from under the skin GIT to the muscles.
الدوالي
Increase in distention of veins spec. femoral vein the valves open & back flow of blood to the lower limb takes place.

The Microcirculation & the Lymphatic System

Each tissue controls it's own blood flow.

Transporting of Nutrients.

Artery must divide many times to enter tissue then divides many times giving Artriols → terminal Arteriols.

Cappillaries.

Artriols → thick muscular coat of smooth muscles.

Metartriols → has muscular coats but not continous.

Metartriols & cappillaries → meet at → Circular forms.
↓ meet ↗ Precapillary Sphincter.

غير عضلي الوريد Venule → can relax & contract but less contractility.

Cappill although muscles are weak but pressure is also low.

Metartrials & Precappillary Sphincter are very near, so their constriction may effect the tissues in supplying the nutrient to tissues.

Passing of nutrients by diffusion or pores.

Types of pores :- thin layer.

1) Intercellular Cleft → containg ridges having pores for passage of substance & linkage.

2) plasmalemmal Vesicle



→ or sometimes these vesicles join together forming (Vesicular Channel.)

exocytosis.

Pores differ from each other in liver, the pores are large to provide passage of protein.

Kidney fenestrated pores. (large). / In brains small pores.

GIT → medium size.

Vasomotion → Intermittent flow sometimes flow, sometimes no. level of O_2 in tissue.

high amount of O_2 is required in active tissue →
He met art~~capp.~~ ride + sp[illegible] ~~[illegible]~~
will open to provide more or close if suffient closes.

(+) Thermal motion through it diffusion occurs.

membrane consist of Lipids → lipid Soluble Subst. can only enter.

Water soluble sub. → channel.

lipids & water soluble → flow in same rate although cleft has small area but it has high rate.

molecular weight $\frac{1}{\alpha}$ diffusion through pores.

that's why not all proteins can enter, albumin the smallest

Nephrotic synchrome → Edema release of all albumin Protein together.

but in Liver all protein can enter for Synthesis.

Water can't pass even if the pores are large, Concen Grad. is very imp.

Starling forces → ① Hydro — except in brain I.f (+)

Interstitial fluid → " — but if (−) it will pull it so I.f.

Cappillary ↑ pressure → flow of blood into Interstitial

Pressure of dissolved protein → Colloidal Osmotic Pressure

① hydrostatic pressure → outside
② Interstitial pressure →
③ Neg. Interstitial press. of cappilary.
} into the cappillary

only Osmotic Press.

Net filtration pressure ⟶ مج. مج.القوى
cappillay pressure ⟶ forces.
from the blood.

Pressure in arterial side is more than the venous End.
as we aproach the vein ⟶ venacava then pressure O:
Osmotic pres. ⟶ no. of particals not the size. الي يهم
fibrinogen large ⟶ but less Osmo. pre.
Albumin ⟶ very small high " "
Small leakage of proteins from blood to I.F.
Incotic p. forms the
The cappillary pressure ⟶ changes & varies. colloid Osmotic pres.
Osmotic p. in plasma & according to the
I.F neg. of I.F ⟶ ثابت Changing in blood pressure.
pressure I.F ⟶ ثابت ⟶ لكن في حالة المرض
(باقي القراءات كلها ثابت)

In the Arterial End ⟶ filteration the ماعدا
While in the venous End, total vol. from diff. 18 ml.
Reabsorption coz the cappillary pressure less
the net (-). while in 10
Sterling ⟶ can know by knowing the (average) Arteriole 30
the diff. between cappillari & I.F pressure. is 0.2 ⟶
In the cases of hypertension ⟶ high cappillary pressure ⟶ 0.2 filtra milli ration.
Edema:

* Lymphatic System ⟶ 1) Immunity
2) transporting Fats, proteins back to blood.
lymph ⟶ lymphocytes taken from nodes, fat, proteins

lymphyated cappillaries → vessels → thoracic ~~duct~~
trunk, Rig. ly. trk

fluid 0.2/min to venules. بيروح

lymphatic cappilleries attached to tissue →
1) anchoring fila-
2) Overlap between edges of 2 lym capp. cells

(mechanism) ← either the ~~two~~ valve pushes the sec. allowing to entring of ~~lym~~ fluid into lymp. vessel but not black flow.

large vessels of veins have many valves.

⊛ Rate of lymph flow :-

① Effect of I.F pressure.

the flow of hight amount of fluid into. I.F high pressure the valve will open & the remaing area of vessel will be compressed due to this pressure. When the 2 forces is at equillibri— opening pressure = compression → stable.

∴ directly propositional untill a stable stage is reached.

⊛ The rate of lymp. flow will increase by increasing I.F (V) — when compression is high constriction of blood.

② Effect of lymphatic pump :-

→ Increasing lymph flow by :- ① Skeletal muscles. ② Artrical pulses ③ Small movements — External factors.

Internal factors. →

distention ال وعاء بعد الملء of lymph vessel وعند constriction ال الماء لا يحدث
ال 2,3 بشكل أكبر

"الدوالي" → Constriction of blood vessel due to presence of specific type of parasite → Viscous Edema.

x

* Blood flow is according to tissue need → Imp.
Liver, Kidney, Brain small portion of body comparing with the whole muscles of the body.
Liver → high metabolic rate.
Brain → CO_2 نسبة ثابتة شرية
if balance become irregular during acidosis, blood high amount is required for clearing CO_2.
Kidney → for excreation & formation of Urine.
muscles → during Excersize → high blood supply.

→ Acute Control for local Blood flow :- 1) metabolic (α) rate. (directly)
2) availability of O_2. ($\frac{1}{\alpha}$)
the lower the O_2 the more the blood flow.
low O_2 → 1) high attitude when travelling from low to high, but people in high att. have more B.V.
2) CN poising. tissue doesnt use O_2 → hypoxia مطلوب زيادة في Blood flow.

* 2 Theories :- 1) Vasodilators → precappillaric Sphincter
يفتح الشريان فيزداد الجريان
→ (Adenosin)
→ hestamin from ATP.
→ CO_2, H ....

2nd Theory :- 1) lack of O_2 → muscles require O_2 for contraction, lack of O_2 prevents contraction so vasodilation takes place direct dilation.

2) Glucose → for formation of ATP.

3) Niacin, riboflavin, Thiamin lack → BeriBeri vasodilation : high amount of Blood from lower part returns to heart → very high contraction → damage death.

* Autoregulation of Blood flow when A.pres. Changes

In acute the flow doesn't return to it's normal but it's better than if pressure ↑ → from perfect posit B. flow into tissue

Myogenic → Stretching كلما زاد الضغط كلما يزيد of smooth muscle → Elasticity → dilation & then contraction وكأنه لم يحدث

Metabolic → pressure ↑ → dilation.

(Micro circulation)

* Reactive hyperymia → flow يزداد ال O_2 بسبب الضغط تمنع مرور لتعويض النقص

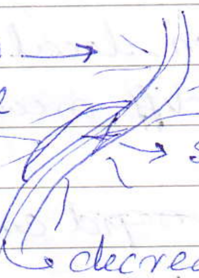
Active hyperymia → Excersize musch → no blocking no decrease in O_2 but the tissue require of O_2 يزداد

when increase in blood flow → Stress on B.V
secretion of Nitric Oxide → dilation of Upstream vessels. to increase perfection of Local B. flow & Efficiency.

Long term control" → long period of time ([illegible]) → but 100 perfect.
reconstruction of vascularity of tissue) → high no of vessels.
high amount of O_2, & dilation takes place.
rate & no. of size & time required for formation of vessels. in neonate high, in old people low. & also in cancer tissue vascularity high + in new tissue such as scar.
but if from high attitude to low → vaso constriction of some extra vessels.

Endothelial growth factor → responsible for formation of vasularization.
disappearce of vessels → Steroid hormon (Cortizol).

Collateral Circulation →



dilation of vessel starts the more the time of blockage the more the vessels opens & Enlarge in size.

The Unsudden block due to age → not very risky doesn't cause angina (partial).
Sudden → Causes Angina.

Humeral control → hormones, chemicals, ions,

CO_2, Hist:.. → vasodilation, K, Mg. → dilation, Lactic acid

Ca, → constritors.

Norepinephrin → Symphethitee nerve

adrenal glend → Norepinephrin.
→ Epinephrin → vaso dilation

Vasopressin → ADH → reabsorption of water.
→ vaso constrictors.

Angiotensin 2 → strong vaso constrictors.
type ②

—x— x — x — x —

Nervous System doesn't control on local tissue but
① Redistribution blood flow to diff. areas of body. more function
② Pumping activity of Heart.
③ Control of B.P → nervous → rapid control
→ hormonal
→ Kidney } long term control.

Autonomic N.S – 1) Sympathetic → heart
→ vessels.
2) para-sym. → heart only.

∴ in vessels only by Inhibition or stimulation of Sympathetic

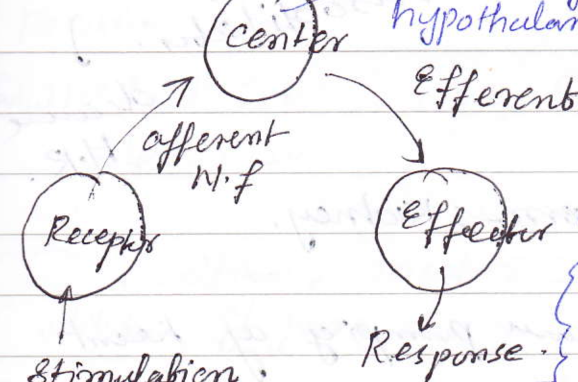
Brain Stem → midbrain
→ pons
→ medule. } Central control of Respiratory circulation.

Sympathetic innervation → ~~force~~ all the circulating structure except metartriole + pre cappillary sphincter + cappillaries.

local [illegible] الأنسجة control.

Increase in the pressure in Artriols, large arteries →
but in Veins high amount of blood
Vaso dilution → heart / due to resistance of muscle

Vaso dilators / Vaso censitor → fibers. mostly found in ~~tissue~~,
mostly. GIT Spleen kidney (Skin) control temp.



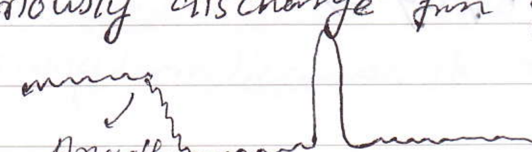
Center → controled by hypothalamus, & Certical area

Efferent { Vasoconstrictor stimul → direct order to peripi Vessels for constrict
Vaso dilator stimul → indirect effect by inhibiting Vaso constriter

diff. between certical area & Vaso center (Vaso motor center)

✓ التحكم بالحلقة عن طريق أوتوماتيكي. مثل إدرار البول (higher center) con

Vasomotor Tone — partically constriction of vessels. ✓ تنفيذ الأوامر only response
continously discharge from Symp.



Vaso constritor area → act on vessels & on heart by increasing H.R
Vasodilator → much less b.y.
Norepinephrin → always vaso constr
epinephrin → vaso dilation in some area & some cases.

Vasodilator → 1) beg. of Excersize.
2 تعمل ايه Vaso dilator في البداية بيدي علشان

2) Emotional fainting (vaso vagal few).
→ Emotional disturbance ⊕ → Ant. hypothalamus.

fainting (Syncope). ← decrease B.P & Blood flow to brain ← Cardioinhibitory area + Vasodilator.] decrease H.R

⊛ Control of Arterial pressure:-
1) Rapid control 2) Long term : Kidney.
by stimulation of Symp. & inhibition of para Symp. → decrease pumping of heart

Total peripheral resistance → only for artrioles highly distributed

muscle Excersize + alarm reaction (fight or flight)

by vaso constriction first to flow blood

Increase muscle Blood flow

local effect + N.S

Vaso dilation at specific areas where blood can flow through it.

Baroreceptors → small bodies in internal carotid & Aortic arch mainly but also in large vessels.
(pressure)
(Stretch receptors)

Stimulated by stretch due to stretch in wall.

كل ما زاد الضغط كل ما زادت ال Signal في ال medula

normal 100 →(+) 120, 130 → maximum

100 → 180, 190 → not maximum (if change occurs near to normal maximum effect).

neg. feedback effect → if increase one factor the other decrease

positive = " → كل ما زاد Contraction يزيد زيادة الضغط في البداية بيزيد بالنهاية. eg. (Pregnancy) ولادة طبيعية

decrease ← stim medulla ← stretch ← زيادة الضغط
dilation of para

In normal condition → when rising from supine to stand position → press decrease due to gravity blood flows down.

2 theories ① Long term ← Baro receptor works to maintain regulation ← no effect due to returning to original (re setting).

② Slow effect in long term, because it inhibits sympathetic stim. in Kidney

Chemoreceptor → when ↓ B.P. ↓ nutrients → ↑ CO_2 / ↑ H^+

only at mean B.P when 80 mmHg.

responded by chemo by ↑ B.P → negative feedback

low pressure receptors →

معدين في أماكن الضغط المنخفض

but not coz they respond to low pressure.

mechanism (stretch) → during I/V drips. (negative feed back)

(Ischemic response)

direct stimulation of Symp.

Indirect stimulant of Symp.

Increase in B. Volume, increase in B.P

Cushioning reflex → Intra cranial hypertension (very hypertension presses on vessels low blood serious supply → decrease nutrients → CO_2 → increase B.P inside the vessels.

if increased in appropriate amount opens the vessels & pressure ↓ but not appropriate very serious vessels remain closed

"Dominant Role of the Kidney in Long Term Regulation of Arterial Pressure & in Hypertension"

— x — x — x — x — x —

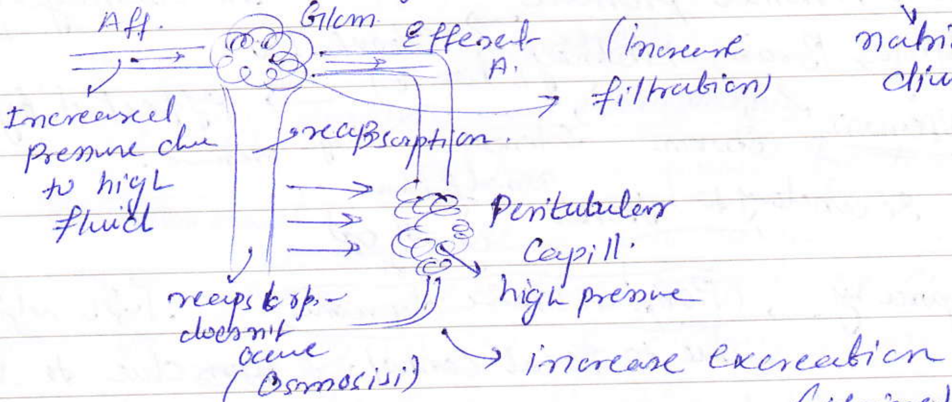
Rapid regu. → fraction of sec to hours.
Intermediate → minutes to days.

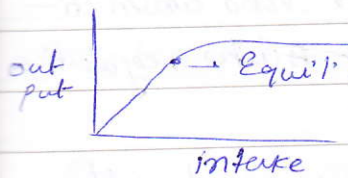
① Renal system → Body Fluid System → also found in some fishes. (primitive).
② Rennin → Angiotensin System.

If Body fluid ↑, B.P ↑, diuresis ↑. / or if there's increase blood pressure the kidney excreation of extra fluid occurs.

natric diuresis → زيادة إدرار الصوديوم في البول

If E.C.F ↑, → Blood Pressure, → Increase water Excreation → pressure diuretics
→ " Salt Excreation → natric diuresis.





→ If Intake increase / output Increase for maintain equilibrium
→ decrease in intake, decrease output.

In cases of diarrhea, dehydration. (heamorrage). ← Retention by Vasopressine. ADH.

E.C.F↑ → Blood pres↑ → Part of it E.C.F.

In some Cases Peripheral resistance may Increase → B.P↑. Out put & excretion Increase to maintain equil. here Cardiac o/put wasn't affected.

Kidney → direct effect
→ or Auto regulation → peripheral resis ↑, constriction.

Hypertension → 1) Salt sensitive hyper. → عالي
2) Salt unsensitive h → because Salt is transported by energy compt while water transfer by osmosis

(*) Volume Loading Hypertension:-
Increase in B.P above the accepted normal range. → The level at which no damaged is done to kidney. mean Arterial pressure → above 110

(Retina, Brain, Kidney, heart) → affected by hypertension
Stenosis, Strokes, Sclerosis, renal failure, high pressure.

Secondary to hypertension

Temporary → (Perip. resistance decrease) ← السبب الا
due to local control. & then due to vaso constriction then Auto regulation
تجربة على الفئران بالإبرة. high pressure

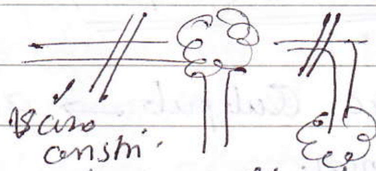
Cardiac out put → عالي
Kidney, pres زيادة، سبب
- resistance
Efferent vessels → juxtaglomerul cells
Gives prorenin → renin → angiotensinogen → ang I → weak vasoconstrictor → ang I (Lung) → ang II
indirectly retension of water, salt; vasoconstric

Pressure زيادة Law: or high (maintenance)

If Shock or heamorage occurs → Nervous system first to act.

Angiotensin acts dire[ctly]

(a) constrict renal ~~...~~ Arteriols



decrease fluid excretion but Increase absorption.
also may be due to (aldosterone) Indirectly.

high amount of Salt eating
Osmolarity increase, thurst for water drinking, increase in volume directly.
Stimulation of hypothalamus by retension.
Vasopressin → ادرار البول

Renal causes of hypertension.

1) Tumer. 2) Patchy ischemia
3) Renal Artery Stenosis.

→ كثرة Renin

when some tissue damage occurs in some places, blood supply decrease renin is secreated

(Converting enzyme Inhibitor)

Essential (primary) hypertension → Idiopathic.

Sec. hyp → caused by deseased.

↓ Increase in Total peripheral pressure without unknown cause.

only Peripl. resistance increase, cardiac output normal.
Cardiac output Increase only in case of Kidney disturbance.

Treatment → By Vasodilators, diuretics, Angiotensine enzyme Inh.

Stress relaxation → Intermediate (delayed compliance).

* Cardiac Output, Venous Return and :-

Cardiac Output → quantity of blood pumped by heart per min.

Stroke Volume → per beat.

C.O = Stroke volume × H.R.

Venous return = Cardiac Output.

Basal body metabolism ∝ Cardiac output.

Excersise ∝ C.O (↓ during rest)

Age $\frac{1}{\alpha}$ C.O

Sex → male more Cardiac O

Basal body metabolism. 5-6 % / 4 - %

due to increase in muscles (decrease with age)

body size → (Cardiac Index) → نصيب كل متر مربع

(according to Surface area).

average:- 3 lit ← for مساحة في الجسم Cardiac output for every m^2.

(body metabolism) → Common factor with excluding other factors.

Young → Small body but high body metabolism.

* Control of Cardiac Output by Venous return.

① Venous Return. → Changing continously. (primary Controler).

② (Heart) pumping. → Permissive Effect

If V. Return was very much the heart stops pumping excess blood.

in normal condition

1) frank-Starling. → changing of stroke vol.

Venous Return → according to the Starling Rule.
increase of Effectivenes of Pumping by changing Venous return, the Stretch will increase pumping.

2) Changing in H.R by. → Stretching of atrium → S.A node increase in it's output > 70-80.
↓ by Brainbridge reflex.
Stimulation by sympathetic to vasomotor.

(Factors):-
tissue metabolism → (∝) Vasodilation → increase in peripheral flow → ↑ venous return → ↑ Cardiac output.

3) Resistance $\frac{1}{\propto}$ venous return:

Cardiac output upper limit → 13 Lit.
physiological limit of normal heart after that plateau.
① hypertrophy → physiological not pathological.
② Stimulation of Sympt. → 30-40 lit by combining both factors.
④ less cardiac output:-
① parasympathetic ② abnormalities.

Right atrial pressure → normaly (0), if pressure increase more than normal → back flow of blood no Cardiac output.
↗ 0 pressure in both areas
but if pressure ↓ → high flow but within physiological limit increase in suction will block large veins entering the chest so will limit inflow. (plateaue).

mean systemic filling pressure → By clamping both sides of Arteries & Veins

Any blockage in Veins is more severe that blocking in Artery due to distensibility & pressure & outflow.

but muscles doesn't undergo vaso constriction → because less amount of O_2 Vasodilation namely due to high metabolism

& In Coronary & brain → no. Constriction.

(powerful venous Constriction).

When only one part of body is exercising & the other is constant higher raise in B.P. due to Vasodilation only in some area while the rest of body vaso constricted so the sum of vaso constricted is higher than vaso dilations.

Coronary circulation → from Aorta → Epicardial / most of Endo. Supply majority of heart, except very few 1% from the blood found in ventricles → Endocard.

⊕ Coronary Circulation → Phasic period during systole & diastole.

direct ⟵ Sympathetic ⟶ norepin.
dilati ⟵ & constrict. ⟶ Epini
but overall constriction by

during systol → less blood because during systo contraction of ventrical, → squeezing of vessels ∴ vaso constriction less blood. & during diastol vise versa of Coronary Art.

& in Blood supply of Artery in Epi cardium less branches than Endo cardium cause contraction occurs from up to down that's why vasoconstriction occurs in Endo so as compensatory End is supplied by more blood branches.

Control of Coronary Blood:-
① Local tissue metabolism.
② nervous system.

Indirect → tissue metabolism → more effective
while direct → end result → because end result
Vasoconstriction.

Ischemic heart disease. → Spasm ~~during~~ in smoking persons.
because vessels are already partially closed.

Acute heart blockage → Sudden death. because no collateral circulation but in chronic, there is time to do collateral circulation

⊛ Distensibility :-

⊛ Note :- distensibility for individual vessel.

بغداد زيادة نسبة الفرق الناتج
diff. between.
Original vol. & the Increased vol. due to increase pressure

flow of blood only during (systol)
no flow but filling during diastole → if no distensibility during diastole → يرجع الـ Vessel لحجمه الأصلي
another push occurs due to returning back from distension :

but due to distensibity flow of blood occurs during systol & diastole.

(1) Accumat ← the pulsatile mot

(2) Incr. of press. ↑ dist. of heart

→ more used than dist. because → for seg.

Compliance or capacitance → المعدل الكمية التي
how much vol. will accumulate in Artery if press. incr. by 1mm Hg. Arterial or Venous syst System

Compliance. → veins ↑. 24 times more
(1) dist ↑, (vol) ↑. arterial & venous (64)%.

sympathetic Stimulation → constriction → press ↑ → but mainly arterial during heme loss of press for
Inhibition. → dilation → press ↓

(Excersize) over all vaso constriction in Venous & Artrial vessels.
no blood loss, veins → heart → cardiac output ↑ →
Arterial pressure ↑ → for supplying so O_2 & nutrition.

→ no relation to symp (normaly). (10 length. bladder ⇄ 20 length)

(1) Delayed compliance :- (1) disappearnce in the desire to pass urine (smooth muscle).
(2) Blood Transfusion.

Sudden Increase or decrease in vol. → Sudden Incr. decrease in pressure due to charac. of smooth muscle. due to change in length of muscle.

Heart failure in old people. very slow to reduce the sudden

By ایلی ایلل ایک ایک Increase in vol. crossing physiological limit → no del. comp.

lower portions of circ → pressure ↓, compliance ↑, P.P ↓, B. flow ↓
no relation with velocity
velocity always with (Cross sect. area).
Aorta → velocity ↑.

Compliance ↑ → syst - dias. → pressure ↓.
cappillaries → no p.p. so that the blood can pass smoothly.
patent ductus Art. → Left. vent. → Aorta → (d.A.) pulm. Art. → lungs → left. vent. → Increase cardiac output (S.V) → Systol ↑ (Sec. circul.) → diastole ↓
(In first circulation) due to transf. of blood from Aorta to P.A.

Mean Arterial press → measuring B.P. for human more than one time in every millisecond for about an hour than we calculate the average, but not as if $\frac{120+80}{2}$ ×

C.V.P → Increase :- 1) Blood Transf. (high volume).
2) less contractility of ventrical, Blood remains in vent. & goes back to Atria & p.a.

contractility/ ~~pressure~~ volume

C.V.P ↓ → ① Haemorrage. ② hypercontractility of Right ventrical → more blood is pumped out → less press in ventrical, all the blood from Atria goes to vent. → P ↓ (less pressure grad

Increase in C.V.P → Increase in peripheral pressure because the pressure in R.A ↑ back-up of pressure into periph. veins → Incre. imp. which leads to open of compressed areas in vein. +4 - +6.

— x — x — x —

⊛ Overview:-

no loss or addition of blood. → any change in a seg. causes change in other seg. (same which is pumped is return)

According to tissue need & activity → during Excersize blood goes to muscles, eating → GIT. that's why after eating no excersize so that blood can flow to muscles.

Content of muscles in wall of (Artery) → great amount of (Elastic) tissue, Strong vessel, Structurally that's why pressure reservoir

Artriole → high amount of (muscles) + Innervation → dilate, constr, control of B.F.

Veins → thin wall → distention. / cappillary → no muscles, one layer of Epi

The velocity decreses in cappillaries → because slow speed is required for exchange of substance efficiently.

Pressure in p. circulation less → 1) because only exposure to alveoli not as in capp.
2) distance is short.

parabolic → centre higher velocity due to less resistance

Obstructed areas → ① Arthrosclerosis.
Turbulence ② Blood clotting. decrease in cross-sec area.
B.F ③ Clossing of valves by back flow of blood from ventricle.

Primary ~~heart~~ hypertension → Increase in resistance in artriole.
(Essential) no cause. (familier tendecy)
(by sec. due to diff. causes).

High viscosity leads to → hypertension

Increase in R.B.C → 1) decreased amount of O_2 ~~in~~ b.
Stimulate Bone marrow to produce more blood. (R.B.C) ↔ { 2) high altitudes 3) Smoking less O_2 in blood, alveoli lined by particles of smoke
4) pregnancy.

W.B.C → less than R.B.C if increase only little except in Leukemia.

Haematocrit → ~~is~~ ~~de~~ can be effected by blood plasma, if plasma decrease H. increase in (concent.) → during dehydration

Sympathetic stim. → Vaso constriction → diameter ↓ → B.F ↓ → more pressure require for pushing blood?.
" Inhi. → Vaso dilati. → diameter ↑ → B.f ↑ → pressure req. ↓.

Pressure in p. circulation less → because only exposure to alveoli
2) distance is short. not as in capp.

parabolic → centre higher velocity due to less resistance

Obstructed areas → ① Arthrosclerosis.
Turbulence ② Blood clotting. decrease in cross-sec area.
B.F ③ Clossing of values by back flow of blood from ventricle.

Primary ~~heart~~ hypertension → Increase in resistance in arteriole.
(Essential) no cause. (familier tendency)
(by sec. due to diff. causes).

high viscosity leads to → hypertension
Increase in R.B.C → 1) decreased amount of O_2 ~~in~~ b.
Stimulate ↔ 2) high altitudes 3) Smoking less O_2 in blood
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→ diameter ↑
→ B.F ↑ → pressure req. ↓.

(*) Rhythemical Excitation:-

Atrial - ventricular muscles → contractile muscles.

Conductive fiber → contracts feebly.

(*) Skeletal musle → no self excitation but by nerves.

but heart → Self excitation in addition to nerves.
System (Autorhythmic)

In normal condition → A·V, Perkinje → Suppressed.

leakage → 1) less neg. of rest pot.
2) abrupt to threshold.

Why the action pot. is less strong (not spike):-

1) Because the [crossed out] volt. req. to reach to threshold is less from -55 → -40

But in V.C → -90 → -40 50 → more voltage, more spike

2) Because no fast Na channels.

Cycle:- leakage → moving the curve to threshould → opening of slow Na-Ca → depol. → open. of slow K → repol. → rep. remain much time (hyperpola) → rebuing the vol. to rest. mem. pot -55.

(*) high conductunce due to → 1) gap junction.
2) size of muscle fiber

bundle of his (Perkinje) → left / right → Endo. → ثلثين ٣
myo. في

Rapid cond in per → Synchronized contraction of vent.

Why the back flow of impulse doesn't occur?

(one direction).

3. Because Refractory period in Cardiac muscle is long, while the conductance by perkinje fibers is very rapid, so backward mov. don't take place because the Cardiac — is still in refractory period when the impulse reaches the end of perkinje fiber.

3. fibrous ~~System~~. Septum.

Sometimes Atrial & vent contracts at same time → abnormal

—— x —— x —— x —— x ——

Circulatory shock & physiology of it's Treatment :-

Shock → Inadequacy of B.F compared with tissue need. not always low cardiac output, sometimes the tissue need may be more than cardiac output.

① Cardiogenic shock → from heart itself.
② hypovolumic shock → heamorrage. (sudden lose, not chronic lose of blood).
③ Neurogenic shock
④ Anaphylactic shock (Allergic shock) → penicillin drug.
⑤ Septic shock (Infection) → Bacteria.

low cardiac output → 1) Cardiac damage not pumping well.
→ 2) decrease venous return.

tissue need Increased → ① Excessive metabolism → Septic shock → hyper thyroid
② Abnormal tissue
Extra vessels, Perfusion pattern
blood goes to more vessels
less blood supply.

Arterial pressure not always low in Shock, sometimes normal.

Stages of Shock :-

① non progressive (compensation) stage :- Recovery occurs.
② progressive stage :- if not treated, death.
③ Irreversable ⟶ no therapy could effect. (no saving of person's life).

Coma ⟶ loss of consciousness but everything may be normal

most common type of shock :- ① Hypovolumic Shock. (decrease in venous return) decrease in out put
⟶ hemorrage.
⟶ loss of Blood (plasma) ⟶ in burning cases ⟶ تسرب البلازما من الجروح
⟶ Severe diarreah, vomiting. loss of fluids & Electrolytes.
⟶ Trauma during accidents (Increase in cappillary permeability

⟶ Blood loss till 10% ⟶ normal.
⟶ without symp. stimulat. & heamorge ⟶ 15-20% ⟶ death.
loss should be (Sudden) ⟶ 20-30 min only.

④ Recovery of hypovolumic :-
① Baroreceptor reflexes.
② CNS ischemic response.
③ Reverse stress relaxation of the circulat. syst (half an hour to hours).
④ Formation of Angiotensin by kidney ⟶ 1-2 days.
Retention of water (Vasoconstriction)

② Formation of Vasopressin (ADH) → hypothalamus.
③ Compensatory mechanisms.
Examples:- 1) Cappilaries → B.P. low during shock reabsorbtion
2) the demand to drink of Blood.
high amount of water.

(negative feedback) (positive feedback) → Uterine contraction.
more till Oxytosine release more.

① Cardiac depression, if blood in coronary artery insufficient
less C.O. → less venous return → damage of muscle of Heart.
② Vasomotor failure → dilation → more C.D.
③ Blockage of very small vessels → normal metabolism.
release Acidic Substances agglutination with occluded R.B.C.
④ Increase cappilary per.
⑤ Released of Toxins. ⑥ Generalized cellular deter.
Endotoxic (by ischemic. tissue. liver, brain, heart.
⑦ Acidosis → normaly Aerobic Oxid.
but in abnormal non-Aerobic → Lactic acidosis → brain.

- Vasomotor failure → Severe less flow of blood,
of tone [illegible] muscles.

neg. feedback → [illegible]
post " → [illegible] Reg.

1) deep general anesthesia → Inhibition of Vasomotor in brain.
2) Sympathetic anesthesia → blockage of Symp. ganglia exsisting from V.C.

(3) Brain damage :-

all venous return low, Cardiac output low ↗ increase capacity of circulation but not meeting Cardiac problem

(4) Anaphylactic Shock :-

Allergic reaction, Antigen + A.B. formation,

Inc permeability ← Stim. ← complex.
of capp. of mast cell in tissue & Basophil in blood.

↓

decrease Cardiac output.

(5) Septic Shock :- if bacteria reaches blood → general Infection

1) High fever → bacterial destruction

2) Vasodilation → by histamin, increase metabolism due to high fever, Infection :- high metabolism due to bacteria require more blood.

3) high Cardiac output due to increase metabolism.

4) Sludging

5) development of Small clots → all over the body.

Treatment :- (1) Replacement therapy :- blood plasma fluids.

dextran → polysac can't exist from vessel (large molecule) can't exist from cell.

Transfusion of blood.

(2) Sympathomimetic drugs → for neurogenic & Anaphylactic
no symp. symp normal.

3) Treatment by head down position. (venous return by gravity).

4) Oxygen Therapy → not very much imp. cause not from lung but from blood.

5) Glucocorticoids → (Stabilizes of lysosomes) stops tissue necrosis
→ met. of glucose.

Circulatory arrest → no consciousness deep comma, but the organs may be normal.

but الموت السريري → الطاقة كلها ع الآلة.

Small clots formation in brain vessels during shock.

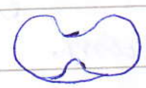
—x—x—x—

Red Blood Cells :-

12/11/013
Wednesday

Total Body fluid :- 42 liters.
↙ Intracellular (28) L
↘ Extracellular (14) L
  ↙ Interstitial 11 L. fluid
  ↘ plasma. 3 l

Blood volume : 5 l
3L → plasma, 2L R.B.C.

R.B.C Biconcave disks 

men : 5,200,000/mm^3.
women : 4,700,000.

, mature R.B.C → no nucleus but cytoplasmic enzymes that can metabolize glucose.

→ normal life span → 120 days.

Haematocrit → percentage of Blood volume that is (cells). R.B.C.
{ 42% male
{ 38 % female.

Function of R.B.C :-

1) Carry Hb which carries O_2 from lungs to tissue.

2) Enzymes called Carbonic anhydrase Catalyse reaction between H_2O & CO_2

$$H_2O + CO_2 \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

3) Transport of CO_2 from tissue to lungs.

4) Excellent acid- base buffer.
Buffer: Substance that ~~combined~~ reversebely with combines hydrogene ions
$Hb^- + H^+ \rightleftharpoons HHb.$

(*) Production of R.B.C in first trimester of pregnancy
R.B.C produced by → Yolk sac — Primitive (nucleated
by in middle trimester → by liver, spleen, & lymph node
mainly.
& at the last month & after
birth by → Bone marrow.

Bone marrow till 5 year produce R.B.C
from 5-20 year → fatty changes in marrow of long bones.
except the proximal portion of Humerous & tibia.
after 20 years the bones can't produce R.B.C. except)
after 20 years the R.B.C produced by membranous bones marrow
vertebrae, Sternum, ribs, Ilium.

(*) Genesis of R.B.C :- Proerythroblast → Basophilic
Erythroblast → Poly chromatophil Erythroblast → Orthochromatic Erythroblast →
Reticulocyte → Erythrocytes.

Reticulocyte stage → contain some basophilic part
in this stage Blood pass from Bone marrow to Blood capillaries
after some days the basophilic part disappears.
& becomes mature Erythrocytes.

If concent. of R.B.C ↑ more than 5,20-...
viscosity ↑, friction ↑ → Blood flow ↓.

Tissue (hypoxia) → cam level to increase R.B.C production.

1) Excessive x-ray treatment destructio of Bone marrow.
2) High altitudes 3) chronic cardiac failure
4) pulmonary diseases. (failure of absorption of O_2). 5) chronic anaemia.

Erythropoytin → hormone directly stimulate the product of R.B.C
90% by kidney / 10% by liver.

Vitamins required for production of R.B.C → ① Cyano cobalamine.
② Folic acid → responsible for DNA production in R.B.C

failure of nuclear division & cell maturation.
cells are larger than normal
Shape oval not biconcave
cells are called primary ← megaloblast.
Cell membranes are (flimsy) (Lio) or fragile & the adult cell → (macrocyte)

Short life span → (megaloblastic anaemia)

Factor secreated by stomach, Specific gastric glands → Intrinsic factor

Intrinsic factor → ① protection of Vit_{B12} from being destroyed by gastric acidity or gastrointestinal enzyme. →

2) Carrying the Vit B_{12} to mucosal cells of Ilium for absorption.

If there's chronic gastritis → atrophy of cells → failure in production of Intrinsic factor → failure in absorption → pernicious anaemia.

normal concent. of Haemoglobin → 12-17 g/dl.

1) 2 succinyl coA + 2 glycine → pyrrole molecule
2) 4 pyrrole → protoporphyrin IX
3) pro + Fe^{++} → heme (Formation of Hb)
4) heme + polypeptide → hemoglobin chain (α or β).
5) 2α chains + 2β chains → hemoglobin A.

Binding O_2 + Fe^{++} → in Hb which is loose & reversable

normal conc. of serum Iron → 50 - 150 micro gram/dl.
(4-5 grams)

Fe^{+2} absorbed by S. Int → in plasma bind with apotransferrin → transferrin → transport Iron to tissue that produce R.B.C

But if there is excess of Iron can be stored in form of (ferretin)

in cells where ~~ferrit~~ iron is stored bind with apo ferretin to form ferritin → in (Liver) or (Reticulo endoth. cells of Bone marrow)
this form of ferritin is reversable when there is need of iron easily could be released, but when there is high excess stored in form of Hemo~~cedrin~~ iron isn't easily released from it.
iron overload → accumation of Hemocedrin in tissues of many organs → pencrease, liver & can damage them. (ceelirin)
usually there's daily loss of iron in Stool → in male 0.6 mg. daily. — Hemochromatosis.
in femele 1.3 mg. due to (menstrual cycle)
The old red B.C become less active & more fragile, they are easily ruptured, fragmentation in Spleen → Hb released
→ Phagocytosis by W.B.C (macrophages) → release of iron ← iron binds with apotransferrin; remaining protoporferin portion of heme → converted by macrophages into billurubin + binds with plasma albumin → hepatocyte absorb the billirubin → in Bile.

Types of Anaemia → Blood lose, hemoragic
1) anaemia → chronic → decrease in iron → ~~macrocytic~~ microcytic hypochromic anaemia.
→ acute
2) megaloblastic. 3) Aplastic anaemia (excessive x-ray treatment) some chemicals & drugs (chloramphenicol A.B.).
4) Heamolytic anaemia → herediitary eg. → Heredity Spherocytosis. cells are very small, spherical, fragile, short life

5) Sickle cell anaemia abnormal Hb → HbS.

damage cell memb. of cell ← precipitation ← low conc. of O_2
Cells more fragile. into crystals. long.

⑥ Pernicious Anaemia:

polycythemia → Increase R.B.C production — Increase in Total Blood volume — Erethemia.

physiological → altitude

Secondary → chronic Cardiac failure.

primary during tumours → polycethemia Vera (genetic abnormality or apperation of Hematopoetic Stem cells that prod. R.B.C). Increase in all type of cells (Increase in Viscosity)

(*) Blood Groups :- mismatch Blood transfusion → transfusion ruption → Immune complex react.

antibodies (Agglutinin) in plasma of one blood reacts with antigen on R.B.C of other Blood.

OAB system → A, B, AB, O.

O → doesn't contain antigen (Agglutinogen).

	A. Bodi.	Anti.ge
O	—	Anti A & Anti B.

O → 47%, A → 41%, B → 9%, AB → 3%.

Immediately after birth → no Agglutinins by 2-8 months prod.

maximum → 8-10 years. then decrease with age.

Transfusion → Agglutination of R.B.C

Antibodies → 2 binding sites or more bind with R.B.C → during few hours to few days after agglutination.

aggl. → hemolysis of Agglutinated R.B.C (delayed destruction).

Sometimes → Immediate hemolysis (less common).

Conc. of B.l Antibodies must be high. → Activation of Complement System.

Type of Antibod.. IgM.

Agglutination of donor R.B.C → because Antibodies of donor blood will be diluted in large plasma of recipient blood.

O → Universal donor, AB → Universal recipient.

(But this not always) → QUIZ. (H.W)

* Rh antigen system:- 6 common type of antigen.

C, (D), E, c, d, e. → most common & most antigenic antigen D.

those who have D → Rh+. 85%.

if absent D → Rh- 15%.

OAB syst → Antibodies are already present, reaction from first exposure.

by in Rh syst → first exposure formation of Antibodies the in sec. transf. occurs.

Sometimes by 2-3 weeks after occurs mild reaction due to less antibod.

↓

multiple Exposure is required to form sufficient A.Bo. for transfusion reaction. →

(1)

Complication of Transfusion → Jaundice increase in billirubine normal 0.3 - 1.3 mg/dl.

Increase in amount of R.B.C → destruction of R.B.C → billirubin.

(2) Renal failure → Toxic subs. from Antigen Antibody reaction & hemolysed R.B.C → occurs renal Vaso constriction & circulatory problems (Shocks) → Arterial hypotension & decrease → renal B. flow → decrease Urine output.

of R.B.C ↑ → Hb ↑,

Small amount of it Hb can bind with plasma protein heptoglobin.

but if excess Hb. → leakage from glumerulus cappillaries to renal tubules. → precipitation of Hb in renal tubule → blockage or obstruction → renal failure.

(*) Erythroblastosis fetalis (Heamolytic disease of new born).

mother → Rh^-, baby → Rh^+.

during the first delivery → transfer of some Rh^+ from fetus to mother blood during placenta separation.

Treatment of Baby → replacement of Blood.

prevention → by giving injection Anti Rh^+ to mother during the first 3 days after delivery.

(Leukocyte & Immunity)

— x — x — x — x —

- Concentration of the diff. W.B.C in the blood :-
- polymorphonuclear neutrophils → 62% } granulocytes.
- " Esonophils → 2.3%. }
- " Basophils → 0.4%. }
- Monocytes → 5.3% / Lymphocyte → 30.0%.) Agranulocytes

→ Leukocytes — main Immunity defence mechanism in body.

granulocyte + monocyte → Bone marrow.

Agranulocyte + lymphs → Spleen, Thymus, Tonsils.

normal range → 3500 - 9000 /mm³.

Neutrophils → mature cells, have the ability to attack & destroy the Invading agents.

monocytes → Immature cells, lesser affinity to bacteria but in tissues enlarge & converted to tissue macrophages. — strong affinity to attack & destroy the bacteria.

Macrophages in liver → Kupper cells.

In subcutanous tissue → histocytes.

Basophil → similar to cells found in pericappillary tissue called mast cells which are larger than Basophil.

both of them secrete → Heparin. (Both of them have relations with Allergic reaction).

A.B → IgE → tendency to attack to mast cells & Basophils → If antigen - Antibody reaction → rupture of mast & Basophils → release of Radicinain, Serotinin, Hestamin & Others ← allergic subst.

Eosonophil → weak as phagocyte, but produced in large no. in patients having parasitic Infection.

(~~Tonsisso...~~). Schistosomiasis → Balarsia.

Eesonophil attach themselves to ~~young~~ form of Schist-
& can kill them. (juvenile)

((Collected in large no. when there's Allergic reactions)).

⊛ Because mast & Basophil cells also secrete (Eosinophil chematotactic factor → migration of Eosonophil to Allergic area → 1) detoxification of some of inflamatory substances, 2) & phagocytosis of Antigen - Antibody compl

((Prevent spread of Inflamentory process))

⊛ Inflamation → when there's tissue damage, the damage tissue release Inflammatory substances which can lead to secondary changes in this tissue → These secondary changes Inflammation. →
1) Vasodilation of local B.V.
2) Increase permeability of cappillary,
3) Increase tissue fluid (Oedema).
4) migration of W.B.C (neutrophils) into this Inflam area.

lekopenia → very low W.B.C produced by Bone marrow. may be due excessive X-ray treatment, gamma radiations, some drugs such as chloramphenicol, thiouracil → (Thyrotoxicosis), Barbiturates.

high amount of W.B.C from Bone marrow → Leukemya. Uncontrolled production of abnormal W.B.C caused by cancerous mutation in mylogenous or Lymphogenous cells, produced in bone marrow

(non-Specific) 1) Phagocytosis of bacteria & other microorganisms invading agents by W.B.C.

2 types of Immunity → 1) Innate Immunity
2) Aquired Immunity

2) gastric acidity & gastro Intestinal enzymes can digest the bacteria swallowed in GIT.

3) Some chemical compounds in blood can destroy bacteria like, lysosyme & Basic polypeptide

Aquired Immunity → cell mediated (T cells).
(Specific) → Humeral (Antibodies) (B-cells)

the Immune System should recognize the specific invading agents by diff antigens.

Specific chemical compound in structure of Invadings which are diff. from other usually proteins or poly saccharides & have high molecular weight.

Specific (T) & B cells → differentiating or preprocessing in thymus glands. in thymus diff, shortly before birth & then for months after birth.

(B) → mid fetal life in (Liver), late fetal life, after birth Bone marrow.

→ during this diff. of lymphocytes develop Extreme diversity → millions of T & B lymph cells that can react against million of (Specific Antigens)

Bacteria phagocytoses by macrophage → release Antigen to T & B-lymphocytes (Activation of clone of lympho.).
(1)
release (Interlokine 1) →
(2)

(*) Activation of B-lympho. → ((differentiation →

Lymphoblast → Plasma blast ↔ Plasma cells → Secrete Anti bodies.

((If subsquent Antigen attacked the person, the secondary A.B respond will be more rapid, powerful & remain for longer time.)) ← if they are not diff. & stopped to develop (memory cells))

(This property was used in cases of Vaccinations))
(period of time between Injections))

Antibodies → 20% of plasma protein
Immunoglobins → IgA, Ig(G) the most in no. 95%.

(*) Action of A.B → (direct)
→ (Indirect)

direct → lysis of Invading Agents, Agglutination, precipitation of toxics. or neutralization.

Indirect → Activation of Complement systems.
(Classical pathway)
↓
11 proteins (proenzyme, enzyme precursor).
($C_1 \rightarrow C_9 + B + D$)

Antigen Anti. reaction
Active site of A.B is activated → activation of C_1
(Cascade of sequential reaction) → multiple End products

multiple End products:-
gives the following reactions:- 1) Lysis 2) Agglutination.
3) neutralization 4) chemotaxis of W.B.C to this area
5) Opsonization:- (Activate phagocytosis of bacteria & other invading agents by W.B.C).

(Indirect).

* Alternate pathway:-
Cell memb. of some bacteria binds with protein B & D. → activation of C_3 → then sequential direct reaction.

* Types of T-cells:- 1) Helpers (the most no. > 75%) & most Imp.
2) Killer cells. 3) Suppressor cells.

Helpers → secrete Lymphokines (Activation of other T-cells & the activation of cells B one of memory).
without the help of these lymp. the immune system become paralysed. thats why AIDS dangerous destruction of Helper cells.

Suppressor cells:
1) Suppression of excessive Immune system to prevent damage of tissue.
2) Role in Immuno tolerence. → during the diff. of (Lymphocytes)
Inhibiting the attack of Immune system against the presence of tissue.
Self destruction of specific T-lymph against the persons own antigens. sometimes some persons loose their immune tolerance → Auto Immune disease → (Rheumatic fever) ← genetic predisposition → upper respiratory tract pre-infection by streptococci → can attack the heart valve & Joints

Some components of toxins released from Strepto coccus.

Cause

1) Acute post Strepto coccal glomerulonephritis.
2) Systemic lupus Erythematosus.

Vaccines → produce Specific Immunity against specific disease by 3 ways:- ① By Injecting bacterias.
Rubel cough, Typhoids.
② Injecting of Toxins → Tetanus.
③ " " Attenuated live microorganisms → but the antigens are correct
(Polyomyelitis, small pox, measles)

Allergy :- genetic predisposition
large amount of Immunoglobulin E.
has the ability to bind to Basophil & mast cells.

Some Allergic reactions
Such as Urticaria → 1) Skin (histamine) Subs. released.
2) Allergic Rhinitis → nose, increase vasodilation → Increase of cap. mucous leakage.
(Histamine).

3) Bronchial Asthma → In bronchioles of lungs (Slow reacting Substance of Anaphylaxes).

4) Anaphylaxes → dangerous, Specific Allergen is injected into circulation
Spasm of smooth muscle of bronchi → difficulty in breathing
Body wide Vasodilation.
leakage of large fluid in Interstitial fluid →

9/dec → [illegible]

(SR.SA)

circulatory shock. (by Hestemin). Or Slow reacting subs. of difficulty in breathing. / Suffocation. Ameyphylexis.

GIT ((Secreations))

(*) Digestive System:- ↗ Parotid, Submandibular & Sublingual but also other glands (buccal glands) can secrete saliva in mucosa of mouth

3 Salivary glands → (Secreation of Saliva).

mucous & Serous secreation (2 types of secreation of saliva).

↓ α amylase enzyme. (Carbohydrates digest). (ptyaline)

Suitable pH → 6-7. for action of enzyme which is swallowed.

Saliva imp. for the health of oral tissues.

1) Washes away the pathogenic bacteria from mouth.

2) " " food particles that could be used by this bacteria.

3) Factors that can destroy bacteria. (such as proteolytic enzymes → lyzozymes & lyses of cell memb. of bact.)

4) Antibodies destroying bacteria.

(Thiocyanate) ions can diffuse to bacteria & kill it. bacteriocydal (kill the bacteria).

(*) Esophagus :- mucous secreation for lubrication & facilitating transport of food.

(*) Gastric Secreations:- 1) Surface mucous cells between glands → Secreate large amount of viscid alkaline mucous secretion for (protection against ① gastric wall & lubrication of food. ②) (continuous layer of)

2) glands of stomach mucosa.

(Oxyntic (gastric) gland & Pyloric gland.)

1) Secreates HCL + Intrinsic factor. by parietal or Oxyntic cells.

2) also secreated pepsinogen by (Cheif) cells or (Peptic) cells)

3) & also mucous by mucous neck cells.

Gastrin hormone has 2 function :- 1) Motility :- Increase pyloric pump.

(2) Secreation :- gastrin act on cells found in Stomach mucosa. (Enterochrom.) which attact to receptr → Secr HCl.

Pyloric gland → large amount of mucous by mucous cell, small amount of pepsinogen by peptic cells + hormone gastrin by gastrin cells.

Stretch of Stomach wall & presence of food release of gastrin hormone → prostalytic movement. (Specialy meat)

but in digestion → cells in Stomach mucosa ← Enterochromaffin like cells. (Stimulated by gastrin)

which secreate histamine → act on receptors found on parital cells → histamine H_2 receptors. → then peritel cells secreate HCl. (called)

during Ulceration high amount of HCl → (Renaticline) Zintac → block the action of histamin on it's receptors (anti hestamin drugs)). (severe gastritis).

pepsinogen —activated by HCl→ Pepsin → suitable pH 1.8 - 3.5. → Protein degradation.

When Stomach is empty → Still gastric secreation occurs Know as Interdigestive secreation less glands mainly mucous & has no acid.

but due to emotional stimuli → increase Interdigestive secreation which will be acidic & peptic → can lead to gastric Ulcers.

HCl → Pepsin.

Pencreatic proteolytic enzymes :- 1) Penc. Amylase → carbohy-
2) Penc. lipase, phospholipase, Cho-
3) trypsin, chemotry, carboxy p.p. → protein. fats. ← Es-
(large compound gland)

⊛ Pencreease :- Endo & Exo crine :-

Exo → acinus glands, secre- digestive enzymes,
Both digest enzyms & Alk. fluid passes together from p. ductule to large P. duct
pencreatic ductuler & ducts → Secreate alkaline fluid.

at papilla of vater : before entering dueden ← Common bile duct ← Pencreatic duct
Surrounded by Sphincter ((Sphincter of Od- control the passage of Alkaline fluid to duo-
open in duodinum

Pencreatic lipase, phospholipase, Cholestrol Estrase, trypsin, Chemotrypsin, carboxy polypeptidase → proteolytic enzymes
(Chemotrypsinogen) procarb-

trypsinogen → (Secreating cells which secreate proteolytic also secreate trypsin inhibitor)). Secreated as inactive because if they are active they will digest the wall of Pencreas
(trypsin) Enterokinase in duoden-
(trypsin → The most imp. & most abundant enzyme by already formed.
other two are also activated by trypsin the more imp.

presence of fats & proteins causes the release cholecystokinin → Secreation of penc enzymes.

Pancreatic alkaline fluid → by Secretin from upper part of S.I in response to acidity of chyme.

N... $NaHCO_3$

Prosecretin → Secretin.
by (acidity) of HCl stomach.

Pancreatic alkaline fluid → (neutralization of acidity) of chyme to protect the (duodenum from damage)

$HCl + NaHCO_3 \rightarrow NaCl + H_2CO_3 \rightarrow H_2O + CO_2$ neutralization.

Inhibit the further activation of pepsin.
Protecting the duodenal mucosa. because only act in acidic medium.
Provide suitable medium for action of pancreatic proteolytic enzymes
pH = 7-8 → neutral to slightly alkali.

Secretion of Bile by liver:-
2 stages :- Initial portion secreted by liver cells (hepatocytes) composed of (Bile acid + lecithin + cholesterol + Billirubin)
Bile ductules & ducts secrete (alkaline fluid) + Bile common bile duct → hepatic duct → Bile either to duodenum or mostly are stored in gall bladder until it's needed by duodenum.

Secretin also stimulate alkaline fluid secreted by bile ductules & ducts.
presence of fat in duodenum secretion of Cholysyst kinin (rhythmical) which causes contraction of gall bladder muscles & relaxing of sphincter of oddi.
Bile salt + lecithin (1) decrease the surface tension of fats & is converted to small particles (fat globules) small size.

Pencreatic proteolytic enzymes :- 1) Penc. Amylase → carboh
2) Penc. lipase, phospholipase, Chol
3) trypsin, chemotry, carboxy p.p. → protein. fats. ← Es
(large compound gland)

* Pencreease :- Endo & Exo crine :-

↳ acinus glands, secreate digestive enzymes, pencreatic ductules & ducts.

Both digest. enzyme & Alk. fluid passes together from p. ductule to large p. duct → Secreate alkaline fluid

at papilla ← before entering duodenum ← Common bile duct ↔ pencreatic duct ← fluid

of Vater: Surrounded by Sphincter ((Sphincter of Oddi
open in duodinum
Control ← the passage of Alkaline fluid

pencreatic lipase, phospholipase, Choleshol Estrase, trypsin, Chemo trypsin, carboxy polypeptidase → to duod

proteolytic enzymes
(Chemotrypsinogen) procarb.

trypsinogen → (Secreating cells which secreate proteolytic also secreate trypsin inhibitor)). They are active
Secreated as inactive because if they will digest the wall of Pencrea

(trypsin) Enterokinase in duodenum

(trypsin → The most imp. & most abundant enzyme
by already formed.

other two are also activated by trypsin the more imp.

presence of fats & proteins causes the release cholecystokinin
Secreation of penc. enzymes.

Pancreatic alkaline fluid → by Secretin. from upper part of S.I in responce to acidity of chyme.

N.. + NG Hcl

Prosecretin → Secretin.
by (acidity) of HCl stomach.

Pancreatic alkaline fluid → (neutralization of acidity) of chyme to (protect) the (duodenum from damage)

$HCl + NaHCO_3 \rightarrow NaCl + H_2CO_3 \rightarrow H_2O + CO_2$ neutralization.

2) Inhibit the further activation of (pepsin). protecting the duodenal mucosa. because only act in acidic medium.

3) Provide Suitable medium for action of proteolytic pancreatic enzymes — neutral to slightly alkaline

* Secretion of Bile by liver:- pH = 7 - 8.
2 Stages :- Initial portion secreted by liver cells (hepatocytes) composed of (Bile acid + lecithin + Cholesterol + Billirubin)
Bile ductules & ducts secrete (alkaline fluid) + Bile → common bile duct → hepatic duct. → Bile either to duodenum.
or mostly are stored in gall bladder until it's needed by duodenum.

Secretin also stimulate alkaline fluid secreted by bile ductules & ducts.

presence of fats in duodenum secretion of (Cholysystokinin) (rhythmical) which causes contraction of gall bladder muscles & relaxing of sphincter of odeli.

Bile salt + lecithin. ① → decrease the surface tension of fats & is converted to small particles. (fat globules) small size.

(*) Functions of Bile (Bile Salt) :-

which facilitate digestion by pancreatic lipase.

Bile salt → (digested fat particles are dissolved in fat soluble ② end products of portions of Bile salt forming → micells ← complexes called (semi soluble in fluids of chyme) as a result (can carry the digestive fat end product to villi where it can be absorbed).

③ Some excess waste products in blood can be excreted in bile. like excess billirubin & cholestrol ... etc.

Bile salts → most of them which are secreted in S.I are reabsorbed 94% → portal blood → Liver cells absorbed & resecreation

Small amount is lost in stool & is resynthesised by (hepatocytes)

(Entero-hepatic Circulation of Bile Salts) → Reabsorbtion of Bile salts

(*) Secretions of Small Intestine :- first few centimetres of duodenum → mucous gland (Brunner gland) extensive.

Secreate alkaline mucous. Sympathetic Stimulation can inhibit Brunner gland.

(3 Sources of alkaline fluid secreation) → for protection of duodenum.

Continous exposure to sympathetic stimulation → acidity ↑ → ulcers. (duodenal ulcers.)

over the entire length of S.I → villi & crypts of Leiberkuhn

In Epith. of crypts & in villi :- 2 types of cells



Goblet cells → mucous / Enterocyte in crypts → Secreate slightly alkaline fluid

While the Enterocytes found in villi.

Enterocyte (paneth cells).. while in villi → reabsorps fluid along with digestive products.

along the villi → microvilli → Brush border enzymes

Intestinal lipase, peptidase → a.a, disaccharidase.
↳ for digest of peptidase

Small

* Large Intestine (Colon) → no villi but Crypts of Leiber Kuhn.

Secreate alkaline mucous. important for

1) protection against (excoriation) due to presence of fibers in stool → friction.

2) provide adhesion material for holding faecal material together.

Alkaline mucous protect Colon mucosa against acidity of Bact activity.

3) Bacteria found in L·I → produce Acidic materials (Bacterial activity).
↓
digestion of small amount of cellulose not imp in humans.
In herbiverous animals. ←

also production of Vitamin K, B_1, B_{12}, B_2 → adva.

Bacteria → (Acidity + gases.) → disadvantage.

(1)

Stomach → normally poor absorptive area, no villi

Junction between Epithelium is tight (2)

most of fluid re absorption in S.I → villi & micro.

Villi → lacteal :- most of fat particles.
lymphatic vessel
↘ Blood cappillaries → the other digestive end products.

large Int :- absorption of (water & Electrolytes) found in faecal material (*) by (Proximal part of L.I.)

only remaining less than 100ml of fluid pass in stool.

23/12/

...resence of water → when moist ...lines resp. pathway, water immed. evap. ...surface of these pathways & humidifies air.

digestive

...lmonary Ventilation :—

— x — x — x — x

Importance or purpose of R. System :— 1) To provide O_2 to tissues & to remove CO_2.

maintain normal conc. of O_2, CO_2 & H ions

The first entrance of Respiratory System :— is (Nose) there are some respiratory function of (nose) :— 1) warming of Inspired air, because nasal mucosa rich in (B.V.)

Contain blood of Body Temp.

(2) Humidification (moistening), related to (vapour) partial pressure) of (H_2O)

evaporation of H_2O from respiratory passage ways, {humidification}

in cold areas, vapour pressure of water → (3.7) mm/Hg

The partial pressure that water molec exert to escape through surface is in Atmospheric air.

(3) Filtration of air :— called → till vap. pressure low. Vap. p. of H_2O in Alveolar air 47 mm Hg

by hair filtration of large particles by reinspired air in respiratory passage way from nose to bronchiole.

layer of mucous + ciliated Epithelium beating towards the pharynx. for trapping of foreign particles.

(4) Other protective mech :—

1) Cough reflex. Bronchi & Trachi (lower respiratory passage) if there's irritating substances this can initiate reflex.

Signals are passed to medulla from medulla signals to cause following events :—

flow of mucous with particles toward the pharynx to be expelled.

a) a large amount of air is inspired 2.5 lit.

b) Approximation of vocal cord. (Shunting).
c) Epiglottis closses opening of larynx.
d) air is intrapped in lungs.
e) contraction of expiratory & abdominal muscles means air intrapped in lungs will be at high pressure.
f) Sudden opening of vocal cord & Epiglottis.
g) expulsion of air strongly outward carrying with it foreign particals.

2) Sneezing reflex:- Same as cough reflex, but it occurs in nasal cavity not in lower resp. passages.
a) depression of uvula. so that the expelled air exit through nasal cavity & not through mouth.
b) lung contraction & Expension (Insp. & Expir).

2 ways of Insp. & Expi:- 1) Contraction & relaxation of diaphragm, decreases the {vertical diameter} or increase of the chest cavity.

2) Elevation & depression of ribs, decrease or increase antero post. diameter of chest cavity.

* Normal quite breathing caused by first way. but heavy breathing ⟶ both ways are participated & muscles - (External Inter costal muscles) & other's Abdominal, Inter Inter costal muscles

((Recoil tendency))

lung → Elastic Structure, has tendency to Collapse.
Complete collapse : expulsion of all air, so that's why there is (force) which maintain the lungs normally expanding. (1)

plueral space → pleural fluid → lubrication +
fluid has (negative) plural pressure which maintain the lungs normally expanded. (2)

→ at beg. of Inspiration this pressure is = (−5 cm of water) Sucking pressure

→ during inspiration → lungs will require more pressure towards neg. → ~~−5cm~~ water.
−7.5 cm

during expiration → neg. decrease again to normal resting level of −5 cm water.

For inward mov. of air from atmosphere to alveoli require

(Boyle's law) ← pressure diff.

Pressure $\frac{1}{\alpha}$ vol.

during inspiration → expansion of alveoli → {vol. increase, pressure decrease}
Pressure diff. → inward mov. of air.

between breathing when atmosp. pressure = pressure in alveoli → 0 pressure
no intake.

Expiration → Compression of alveoli → vol↓, pres↑, movement toward Atmosphere.

Cause of (Recoil) tendency :- 1) The elastic (force) of lung tissue itself

2) Surface tension of fluid lining, alveoli & other lung inside wall air spaces.

2/3 of Recoil tendency → due to surface tension of fluid.

(Type II Alveolar cells)

Special Alveolar Epithelial cells :- Secretes substance called pulmonary (Surfactant) → decrease the surface tension.

In Embryonic life :- Secreation of surfactant from 6-7 month. Sometimes later.

That's why problem may occur in premature babies.

← Causing (Respiratory Distress Syndrome of newborn))

— × — × —

Definitions of pul. (volumes) & (Capacities) :-

1) Tidal volume :- Vol. of air inspired or expired with each normal breath = (500) ml.

2) (Functional residual capacity) :- amount of air remaining in lungs at end of normal Expi- = (2300) ml.

But in gaseous exchange site → 350 ml of (Tidal vol.)
Alveoli, Alveoli ducts, Alveolus Sacs

The remaining vol. = 150 ml in areas where is no gaseous Exchange → dead space

(dead space air) → Nose, trachea, pharynx. from bronchi upward.

1)

The rate at which new air reaches the gaseous area is called A.V

{Alveolar Ventilation} time (rate) → 1) Amount of air reach the gaseous exch. Knowing × alveolar resp. per/min

350 × 12 = 4200 ml/min.

1- alveolar Epith.
2- Intsa Space
3- capp. memb.
4-

6 Comp. of alveolar resp. membrane → where gaseous exchange occurs
?? why this only

0.6 micrometer.
= 1/million

(*) Diffusion of O_2 & CO_2 Through Respiratory memb.
factors :- 1) pressure diff. (alveolus → pul. capillary) O_2 ← CO_2
depending on {partial pressure}

2) Thickness of respiratory membrane ∝ 1/ to diffusion.
① (Edema fluid) thickness ↑.
causes ✓ → of Increasing thickness.
② Some pul. disease can cause fibroses increase thickness of resp memb.

3) Surface area of Respiratory memb. ∝ diffusion.
eg. 1) Removal of part of Resp memb.
2) pulmonary Emphysema (dissolution of alveolar wall). decreasing the S.A. { excess air in lungs.

4) diffusion Coeffiencient of gas ∝ $\frac{S}{\sqrt{MW}}$
S → solubility of gas
MW → mol. weight of gas.

Pulmonary Odema, Pleural Effusion, Pul. Emphysema

diff. coefficient of $CO_2 \rightarrow$ (20) → O_2 سے 20 گنا
$O_2 \rightarrow 1$
(rate of diff. CO_2 more rapid).

1) Part. p. PO_2 in pul. art of syst vein 40mm Hg. after gases exchange 104. in pul. veins

partial pressure of O_2 in alveolar = (104 mm).
Arterial end = 40
Venous End → 104.

diffusion of O_2 from tissue cappi to the cells.
Arterial End of cap. (Tissue cells) venous end (40) mm Hg. Vein
95 mm Hg
Art

lungs میں ویسے ہی

2) In tissue.
$PO_2 \rightarrow 95$ mmHg
Bronchial art. Blood sup. to lungs. & Bron. vein into pulmo. vein

uptake of CO_2 by blood tissue capill
Art end → 40 mm Hg. (Tissue cells 46.) 45 mm Hg

O_2 conc in Bronchial vein 40 & into pul. vein which have conc of (104).

diff of CO_2 from p. Blood into alveoli.
Alveolus → 40
A. End → 45 mmHg
Venous end = 40.

الفا کی جگہ میں 2300
with each breathing not complete
renewal of air, but partial (850) ml from (2300) ml
means (1/7) of air in alveoli)
because if complete Imp. replacement leads to sudden & rapid change in gas concent. concentration in lung.
Partial renewal of air
(Slow renewal is Imp.)

95 اور 104 میں O_2 کی کمی ← (Venous admixture of Blood)

↗ 0.3 ↗ 40

Atmospheric air is not equal to Alveolar air →
diff. بسبب O_2 اللي اتخدت due to partial of O_2.

due to absorption of O_2, renewal of air.

CO_2 → تزداد — exchange of CO_2.

H_2O → لا تتغير بسبب
Humidification.

« Skin and Sense Organs »

—x—x—x—x—x—

→ Epidermis, dermis, Hair structure, Hair follicle, glands of the Skin, Sebaceous & Sweat.

→ Eye – Structure of Retina.

→ Ear „ Internal Ear.

Integumentary system formed by :- Skin or cutaneous memb.
Body's biggest & largest organ, first Line of defence, total area of 1.5 to 2 m². about 15% of total body weight

Appendages → Accessory Structure

* Skin functions :- 1) protection 2) Sensation 3) Thermo regulation
  - drying out water & Air by creatine.
  - from microorga by physical barrier.
  - → from Sun UV light
  - → from Injury

* Sensation → Touch, pressure, pain & temp.
→ Thermoregulation. → role of surface vessels, sweat gland. decrease the heat.
→ Metabolic function.

Structure → 2 layers, depend on tissue type, thickness, vasculatory, or Embr origin & function.

first layer (outermost) → less thicker avascular → Epidermis. → Ectodermal in origin.
funct → protection (melanin - creatine). (Stratified squamous keratinized).

Sec layer (dermis) → (thicker) → vascular → mesodermal in origin
Support & nourishment (Lymph, nerve).

Hypodermis → Superficial fascia, no skin layer
loose areolar C.T, binds skin to adjacent organ.

Langerhans, keratinocytes, melanocytes → Epi.
5 layers structuraly diff layers. → basalis, spinosum, granulosum. ..

2 types of skin depend on no. of Epidermis.
Epidermis → non constant layer, dermis → constant layer.
most of body covered by thin skin, except palm, fingers, toes, sole feet → thick skin.
thin skin → 4 layers comparing with thick skin.

, means layer rest of name according to morphology or function

Stratum basalis, Spinosum, granulosum...

(1) Stratum basalis → Simple cuboidal Epith., lot of mem nucleus single, cyto. → basophilic, high metabolic activity to give new cells for superficial layer. Inter. keratine layer.

(2) " Spinosum → prickle cell 8-10 cells, longer than cell, .. (Spine projections)

(3) " Granulosum → have granules multiple 3-5, each cell nucl. flat; cyto: - basophilic (lot of granules)

(4) " Lucidum → found only in thick skin. (clear) Clear 3-5 cells, keratinocytes parallelly nucleus → not seen, cyto filled with eledin.

(5) " Corneum → superficial of Epi, horny cell layer. dead keratinocytes cells increase thickness. Cells → Squamous.

* Keratocytes → keratin producing for protection gives hardness.

desquamation → process of cell shedding from cornium.

Keratinization → ... replacement.

Langerhan → (Immune cell), phagocytose, derived from bone marrow.

Merkel cell → touch receptors in basalis, from neural crest, finger tips, soles, makes synaptic Junctions. cyto:- small dense granules.

(*) Skin colour :- depend on pigmentation. → dependent. 1) melanocytes.
Originally skin colour → 2) B. vessels: (Hb).
yellow, but due to blood 3) ~~Carotin~~. ~~Carotin~~. (Carotene)
vessels dark colour.
Carotein → accumulate in Epidermis + fatty tissues.

(*) Melanocytes → deepest basal layer, secretions to superficial layer to appears skin colour.
cyto → melanin (melanosomes).
Epidermal melanin Unit → no of melanocyte per unit.

melanocytes in white people → in Stratum basalis.
but in black people melanocyte → reach Spinosum.

Melanosomes → temporary structures.

not to diff. no. of melanocyte but diff concentrat of melanin → Skin colour.

→ heredity defect.

⊛ Albinism → normal melanocytes but not melanin, abscence of tyrosinase enzyme. depegmintation,

⊛ Vitiligo → no melanocytes.

⊛ Dermal - Epidermal Junctions → ~~hemosomes~~ desmosomes, hemidesmosomes.

⊛ Finger print:- Ridges in palm & sole deep.
→ to increase friction & S. Area of skin.
→ protection.

⊛ Dermis → dense irregular, underlying C.T mesodermal in origin.

2 layer of CT proper → can diff. by forms. also contains Elastic & collagen fiber

Super layer → dermal (papillary layer) → Invagination of layer by through. bush.
(loose) → more B.V

thin layer → loose areolar C.T.

(deep reticular layer) → dense irregular, more fibers, fewer cells.
↓
less B.V.
fibers arranged in diff direction.

⊛ presence of Elastic System → Strength, Stretch, return to shape). (مطاطية الجلد)

Thick skin → no hair follicles.

Skin → most Extensive sensory receptor. Send impulses to spinal cord.

1) thermo receptor 2) Meissner's

Epidermis → free nerve Ending

dermis → subcut tissue (encapsulate & Expand receptors).

Hair → Elongated Keratinized Epith. thread

face → 600 hairs/cm², remaining → 60.

Hair growth → affected by hormones.

(prevent heat loss, UV protection.)

→ hair follicle → (cortex, medulla, cortical area of hair).

around this 2 sheets → External ~~root~~. root
→ Internal ~~root~~. root.

Hair shaft, Hair root, Hair bulb.

root → at the base of Hair.

hair arise from Epidermis, dermal papillae CT in lower part of hair.

glassy memb → non cellular part, ~~xxxx~~ hyalin part sep. dermis from Epi.

* Arrector pilli muscles → Erection of hairs when contracted.

* Sebaceous Oily gland.

Skin → mirror reflect your health condition

Psoriasis → Silver, Scaly skin, increase mitoses, increase Epidermal thickness.

(*) **Eye** :- Complex, photosensitive.

Element of Retina → pigment cells, photoreceptors.

→ Layers of Retina :- (10 layers) ① pigment Epithelium. ② photoreceptors, ③ External

(Ear or Vestibulocochlear Apparatus)
(Structure of gones / Equilibrium.

$CO_2\uparrow, H^+\uparrow$

Regulation of Respiration:-

Respiratory centers:- in medulla

1) Dorsal Respi. group.
2) Ventral " "
3) pnemotaxi center.

1) Dorsal → transmits (Insp. signals) repetitive action pot, which begins weakly & increase gradually for 2 sec. then stops for 3 sec (when Expiration occurs)

In order to :- «Ramp Singnal». in normal quite respirati.)
(Steady increase in Volume of Lungs.) & not gasping as in some animals.

nervous signals → these signals goes to diaphram causing normal quite respiration. → Expiration in normal case passive process, relaxation of diaphram & Elastic recoil of chest & lung.

(2) pnemotaxic → sends inhibitory signals to dorsal Respi. group, to control (Switch off point) of ~~Expiration~~ Inspiration (Insp. ramp signal) → after 2 sec if pneum. sig. was normally normal.

$\frac{60 \text{ sec}}{2+3} = 12$ → normal respirat. rate /min.

2+3 sec → Expiration
↓ Pnem. Imp.

(α) But if pnemotaxic impulse longer strong. respiratory rate increase $\frac{60}{3} = 20$ ↑

pnemotaxic center & (Vise Versa). → eg. $\frac{60}{4+3} =$ ↓ i.e.

limits duration of Inspiration & Increase respiratory rate.
Because limiting Inspir. also shortens Exp. & the entire respir. process.

Exp. → Normal Elastic recoiling of lungs.

In normal case quite breathing due to repetitive Insp. signals from Dorsal R. group to Insp. muscles mainly diaphragm

(3) Ventral → in normal quite respir → Inactive but in heavy breathing → dorsal group send excitatory signal to ventral. → send excitatory signal to inspiratory & expiratory muscles.
Contribute further in Respiration in heavy breathing.

(*) Protective Reflex (Hering–Breuer inflation reflex). → in wall of Bronchi & Bronchiole → Stretch receptors. If tidal vol increase above 1.8 half vol. → over stretch. of lungs, stretch receptors send inhibitory signal to dorsal group, prevent further inflation of lungs.

(*) Chemosensitive Area. → main stimulus H^+ ions. (medulla) (CO_2 little).
(Excitatory signals to dorsal group)
But person who have CO_2↑ → Respiration increases.

In person who have H^+ much → H^+ doesn't pass easily through Blood brain barrier but CO_2 passes very easily thus person with high conc. of CO_2 increase respiration

$CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow HCO_3 + H^+$

This chemosensetive area in 2 days → occurs adaptation.

decrease. → not to normal but toward normal. عكس التأثير الأولي التأثير يصبح & Ht of initial Effect

H^+ become toward normal ← Blood HCO_3 تمكن بيزداد due to {renal regulation of Acid-Base ↔ Balance.}

{CO_2 → Effect central & peripheral}.
but doesn't effect respiratory syst. clinically.

decrease in O_2 → Stimulation of {peripheral chemoreceptors in Carotid body, few in Aortic) bodies, very few in thoracic & abdominal arteries. (mostly in bifurcation of com. carotid — receives rich blood supply with O_2)

if less O_2 Excitatory signals to dorsal group. (also increase in CO_2 & H^+ but less. ← stimulati for perip. chem. but CO_2 effect centrally less.)

Effects of CO_2 in peripheral more quick than central but in central is much in quantity → {Imp. in onset of Exercise}
CO_2 has effect on central (medulla) & peripheral chemo. is then.

dorsal → Increase respiration as a result CO_2 & H^+ decrease a little causing {relative depressive effect on Respiratory centre}
وبالتالي ؟ تأثير نقص الأكسجين المؤدي إلى زيادة التنفس ليس كبيراً بسبب
لا يحدث الزيادة في التنفس بشكل كبير.

But If persons climbs mountain at the peak → low O_2
If climbing during hours → respiration increase only by 70%.
after 2-3 days relative depressive effect occurs on Re. cent. (adaptation)

وبالتالي لا يتأثر respir نقص الأكسجين وبالتالي تزداد respiration (400) (500) times
& CO_2 & H^+ syst.

Acute low oxygen concenc. → increase in respiration is low.
but if chronic " decrease " → Increase in respir. high ↓
هذا ما يحدث في حالة السكان في أعالي الجبال

Phenomenon of {Acclimatization}
(ظاهرة التأقلم)

Rapid response of per. to incr in CO_2 during Exer.

Blood. ← كنترول الإدراك

digestion. ← الشبع

Imp.

{ climbing Exercise motor cortex, of brain, send exsitatly signals to muscles & send collateral sig. to resp. syst to increase respiration. (but less effect)

also chemical factors → {fined adjusment of respiration to maintain proper} con. of CO_2, H^+, O_2 in tissues during Exercise

[illegible]

CO poisoning

↳ affinity to Hb like O_2 or faster. / bind to iron, dis place to O_2.

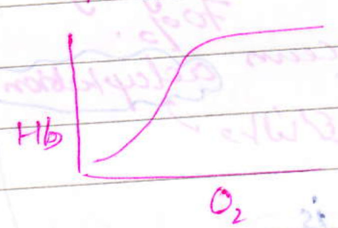
If concen. of O_2 → 95 %. mm mercury.

& concen. of CO_2 → 0.4 mm mercury

Hb binds to CO more than 90%. (very dang.).

CO → Colorless & Odorless.

Binding of Hb with CO → keeps the dissociation curve of O_2 / Hb shift to left, decreases the release of O_2 to tissue. release decrease & binding increase.



Main characteristics of Leukocytes :-

(1) Classification 7 types of L. (Granulocyte / Agranulocyte)

(2) They are involved in Allergic reaction + Inflamatory reaction

(3) Imp. defence mechan. as primary defence line.

(4) Responsible for Immunity (Lymphocytes, Antibodies)

—x—x—x—x—

Digestive System (GIT Movements) :-

—x—x—x—x—x—x—

GIT smooth muscle is considered as 'Visceral Smooth muscle' or known as ((~~Sensitized~~ smooth Synsitial muscle))

Smooth m. fibers are connected by gap junctions in which movement of ions is slow... So if Initiation of A.P occur in any point. A.P can spread to whole muscle bundle, → thus muscle contraction occur very quickly.

~~[illegible]~~ Resting memb. of smooth visceral muscle → -65 mV in slow waves (changing in resting membrane potential) when resting memb. pot. reach the Threshold = -40 for initiation of A.P. known as (Spike potential).

Many factors do depolarization of GIT smooth muscle :- (Stimulation)

(1) Stretch of the muscle. (2) Para Sympathetic Stimulation with secreted Acetyl choline.

(3) Stimulation by some GI hormones.

(*) Inhibition or (hyperpolarization) of GI S.M:-

1) Sympethetic Stimulation → Epinephrin some times & Norepinephrin.

(2) GIT has special nervous system called → Enteric N.S.
Enteric N.S extends from Eosophagus to Anus & is composed of 2 plexus:- 1) Mesenteric plexus → GI movement control
2) Submucosal (Meissner's) plexus → (GI Secretion & local Blood flow control)

Autonomic Nervous System (symp. & para.) has connections with Enteric N.system.
In general parasymp stim. will increase GI activity while symp. stim. inhibit GI activity (also include secretions).

(*) Basic propulsive movement of GIT → Peristalsis

The usual stimulus of peristalysis is → distention (1) — Inherent property in many sensitive S.M tubes like GI tract, Ureter, fine ducts & others.
If any point in GI tract occur distention, 2 cm behind the site, occurs constrictive ring & this ring then spreads along the tube forward in anal direction of distension.

(2) Other stimuli → parasymp. stimulation or irritation in Epithilium can also initiate parastylasis. (3)

towards the anus
receptive
everal cms forward or down stream occurs → relaxation
help in propulsion of food during propulsion of food.
to stomach. (facilitate food propulsion)

fter Chewing, swallowing of food occurs.
wallowing mechanism has 3 stages.
① Voluntary stage   2,3 → Involuntary (pharyngeal & Esophageal).
The mm pushes bolus of food post. by tongue towards the pharynx.

2- Pharyngeal stage → when food reach beg. of pharynx
Excitation of [swallowing receptors] which sends signals to
the swallowing centers in brain stem which sends impulses
to cause the (following automatic muscle reactions.) –

[1] Soft palate is pulled upward to close the posterior nares
to prevent reflex of food into nasal cavity of nose.

[2] Palatopharyngeal of folds on each side of pharynx
are pulled medially forming small slit, through which
well masticated food can pass.

[3] Larynx is pulled upward & anteriorly (The vocal cords are Approximated)
& Epiglossis closes opening of Larynx. to prevent passage of food to Larynx & then to trachea.

[4] upper Eosophageal Sphincter releases, Sphincter usually tonic contraction (prevent passage of air to oesophagus ~~during~~ respi).
Stomach.

[5] upper part of pharynx begins peristalytic waves for propulsion of food to Eosophagus.

All these occurs in less than 2 sec & withing this stage occurs inhibition of respiration because swallowing centre sends signals to inhibit respiratory center.

⊕ Eosophageal Stages 1) Primary peristalysis :- continuation of peristalysis that begun in pharynx.

2) Secondary peris. → If primary peri. doesnt propels the food.
↓
Stimulated by retention of food → propulsion of remaining food into Stomach.

(Cardiac Sphincter)
In Stomach → Gastro Esophageal Sphincter (Tonic constriction). prevent reflect of gastric secreation to Eosophagus (To prevent damage of Esoph. by acidic subs).

during coughing, heavy breathing or walking → increase in intraabdominal pressure → Increase in Intra gastric pressure.

that extends to stomach

may cause reflux of gastric contents to Esophagus, but the short portion of distal part of Esophagus occurs causing causing valve like (end) closure. preventing the (inward reflux of gastric secretions)

When food is in stomach 2 activity :- ① Mixing contraction
② propulsive contraction

most of time when food in stomach → (Mixing contraction) or (weak peristaltic mov.) → To mix the food with gastric secretions

In pylorus → pyloric sphincter :- prevent propulsion of food from stomach to duod. during contraction but allow the passage of fluid.

After that these contraction become strong → pumping of food from stomach to duodenum causing → {Pyloric Pump.}

factors increasing pyloric pump (Gastric factors):-

① Increase food volume (Stretch of stomach wall → stimulation of Myentric plexus → Increase pyloric pump & relaxation of pyloric sphincter.

② Hormone (gastrin) → stretch of stomach wall together with some digestive products specially meat → gastric gland presence secret gastrin → Increase pyloric pump.

Stimulation of myenteric plexus
decrease Pyloric pump. → Contraction of P.S

(Duodenal factors) Inhibiting pyloric pump (Entero gastric reflex).

(1) Excessive distention of duodenum.
(2) presence of Irritation of duodenal mucosa (Chemical substance) or presence of excess acid in chyle. (Enough time to make neutralization)

(3) Excessive digestive products of proteins. & some fats. (Enough time to provide for it's digestion)

Osmolarity → Total concen. of solutes (regardless their exact composition) in a solution

(4) presence of Hypertonic or Hypotonic fluid.
Osmolarity may be high or low.
Normal Osmolarity → (300) mOsm/L.
because if rapid absorption of Hyper or hypo tonic fluid occurs this result in rapid change in Electrolyte concentration of Extra cellular fluid.

(5) presence of fatty food in duodenum (upper part of S.I) → release of hormone (Cholysyctokinine) → Inhibition of Pyloric pump by inhibiting action of Gastrin.

→ Mixing Cont. in Small Int. is diff:- (Special chances).
when portion of S.I is distended → localised contraction spaced at Intervals → known as Segmentation contraction.

when a set of Segmentati - contract relax, new sets occurs at new points between the previous contractions → (helps in multiple forward propulsion of food (chyme))

peristalysis contract in S.I (Seg. contr.) → (Benifits) ① mixing of chyme with S.I secr. , ② Slow propulsion of chyme.

but prop. contrac in S.I very weak → mov. of chyme in S.I is slow → To have sufficent time for digestion & absorption.

At the end of Ilium → Ilio cecal valve → prevents back flow of fecal material from colon to S.I.
Ilio cecal Sphincter → normally mild (tonic contraction)
it slows entering of Ileal contence to colon, presence to facilitate more absorption

"Function of Colon:" ① Absorption of H_2O & Electrolytes.
② Storage of fecal material until it can be expelled.

proximal half of colon concerned mainly with → absorption
distal " " " " " → Storage.

On colon :- 1) Mixing contraction → 1) Hostral contractions
mixing of fecal mat., facilitate absorption of H_2O & Elec.
2 - Slow propulsion of fecal material
anal wall
Specialy in cecum & ascending colon
2) propuls. contr.

(4) Modified type of perist. begins in Trans. colon

When (Irritation) in any point in colon spe. Trans. colon. (Mass movements)
(Constrictive ring) appears, then (20) cm or more distal to this point (contract unit) occurs forcing the fecal matter towards the rectum, when fecal mat. enters the rectum → the (desired for defecation occurs) →
by mass mov.

occurs defecation reflex. (pers. waves)

Which includes:-
{Contraction of Rectum
& relation of Anal sphincter}

2 types of Anal sphi. → (1) (Internal) (Smooth mus).
Involuntary sphincter.
(2) External (Straight muscle) → Under volun. control
Voluntary relaxes Anal External sphincter.

—x—x—x—x—

Components of defecation reflex.

but CO_2 is permeable to B.B.B

Chemosensitive area → Is sensitive to high concen of H^+ & less to CO_2, but CO_2 indirectly effect this area by changing concen of H^+, because H^+ can't pass through Blood Brain barrier, thus when CO_2 concen. increase in blood, so thus in cerebrospinal fluid CO_2 ↑, so CO_2 in this fluid react immediatly with H_2O to form H^+ & much amount of H^+ is released to the chemo sensitive area. more H^+ is released to this area when CO_2 ↑, then when H^+ in blood Increase

but after 1-2 days adaptation occurs, & CO_2 no more has strong effect due to renal regulation release high amount of HCO_3 which binds with H^+ decreasing it's effect.

(*) Transport of O_2 in Blood in form Hb by binding to
(1) Hb 97% O_2 in blood by binding with (Fe) portion of Hb, this binding is loose & reversable (2) the remaining of transport of O_2 is in dissolved state in plasma 3%.

(*) O_2/Hb dissociation curve: when pressure of O_2 ↑, the percentage of Hb bound to O_2 ↑ in blood.

eg. if pO_2 → 95 Percentage of O_2 bound to Hb 97%
Systemic artery. pH

if pO_2 → 40. per. of Hb bound to O_2 → 95%
in syst. vein

كلما زاد press يزداد (of perc. Hb bound to O_2. & vise versa) العلاقة طردية الخطي ليس

(*) Shift of curve to right → when there's increase in CO_2 as a result increase in H+ concen. or increase Temp. (because HCO_3 carbonic acid ↑) all these shift the curve to right → which means decrease binding with O_2
If shift to left → Binding increase.
In tissue capillary → Shift of curve to right because more CO_2 will pass to capillary → H+ ↑, as a result more O_2 will pass to tissue cell.

Normal = 7.4 7.2

but in lungs → Shift to left, because more CO_2 will pass to alveoli → CO_2 ↓, Blood will become less acidic or more alkaline.
(binding w.r.t. of O_2 ↑.)

↓ this effect — Thus pH = 7.6.

(Bohr Effect)

during Excersize shift to → right, more CO_2 ↑, acidic subsc. release, temp. of muscles increase. H^+↑
Binding with O_2 ↓, more release of O_2

Increase in BPG - 2,3, Biph——— Phosphate compound normaly in Blood, which shift curves slightly to right.
But in hypoxic condition, BPG Conce increase, thus more shift of curve to right → release more O_2 to tissue cell. Acidity ↑

Haldane Effect → Binding of O_2 with Hb tend to displace CO_2 from blood.

① { makes Hb more acidic
as a result the more acidic Hb has less tendency to bind with $[CO_2]$.

② More acidic Hb has tendency to release H^+, which binds with HCO_3

converted carbonic acid

$CO_2 + H_2O$ → occurs in pulmonary capp. pulmonary cappillary.

$H^+ + HCO_3 \rightarrow HCO_3 \rightarrow CO_2 + H_2O$

But in tissue cappillary → the less aerobic Hb has more tendency to bind with CO_2, thus more pick up of CO_2 in tissue.

(*) Transport of CO_2 in Blood → 1) (7%) in dissolved State
2) by binding with Hb forming {Carbamino Hb.}. $Hgb \cdot CO_2$ (loose & reversable). about 20%
3) also small amount of CO_2 can bind with plasma protein. (23%) 3%.
4) The most imp., in form of Bicarbonate → 70%.

$$CO_2 + H_2O \underset{\{Carbonic\ anhydrase\}}{\overset{Transp.}{\rightleftharpoons}} HCO_3^- + H^+ \text{ (acid)}$$

Respiratory Unit → Resp. Bronchiole, alveolar ducts, Atria, alveoli.

Rep. memb → page (497).

Venous admixture of Blood →

Pul. Alveolar PCO_2 = 104.

40mmHg − 104 − (104)

Pul. Artery. Systemic Pressure — gaseous exchange — Pulm vein.

∴ pul vein = (104).

Arterial end 40 — 40 venous end — Syst. vein

(95) (23) Tissue capp.

Shunt Blood.

reason of 95 → 98% of Blood reaching left Atrium
Oxygenated through (Alveolus) & thus → 104.
2% → ~~or~~ leaves the ~~...~~ heart in Aorta & goes to
Bronchial circulation supplying deep tissues of lung
without passing through (Alveolus) thus when leaving
lungs → (40). when this Blood of Bronchioles (40)
mixes with Blood in/from pulmonary vein (104) → this
effect ~~effect~~ causes decrease in PO_2 to 95 mmHg.
{Venous admixture of Blood}.

CO_2 →

Alv PCO_2 (40)

Pul. Art. — 45 — (40) — 40 — Pul. veins

Tissue capp.

Art. — 40 — (46) 45 — 45 → Veins.

during Excessive Skeletal muscles

vasodilation of Arteriol occurs → decrease in T.P.R. →

Const. ← Stimulation ← Decrease in
of large of Vasoconstrictor B.P
veins centre in
↓ brain

blood flow to Heart ↑ → Blood press ↑.

In persons having (hyper tension) → capp. pressure ↑, high filtration ↑, Odema if no lymp. syst.

{ lymph. cappillaries. → lymp. vessels. → Thoracic duct.
→ Right lymphatic duct }.
(Charact of each. type.)

1) Anchoring fil.
2) Overlapping betw 2 adjacent End. cells forming valves.

lymph vessels → enters the lymph nodes.
(valves) in one direction.

(Blood pressure↑ increase the rate of lymp. flow)

The diff. between the fluid in Elephantiasis & Odema.
due to cardio.. in Elep. → milky appearance of fluid due to lymph.

(1) Rate of diffusion of lipid sol. subs. = rate of diff. of lipid insol. through pores.

(2) Mole. size of particles $\frac{1}{\alpha}$ rate of diff. through the pores only.
In muscles → not all proteins can pass.
only albumin (very small) can pass very slowly.
Nephrotic Syndrome → all albumin released, releasing with it water & fluids causing swelling, Odema.
liver → large pores.

Conc. grad. → very imp. even if pores are large.
if Con. grad. equal no net moveme

pores → Concen. grad.
pores → Mol. size

pressure always → pushing force in opposite direct
except in case of (−) Int. fluid pre.
In normal condition → Int. fluid always (−)
except in some places.

Osmotic → no. of particles no size, albumin small more amount 80% resp. for Osmotic presse.
small leakage of Albumin 1/1000 into interstitial fluid.

1/10/

...ation → Exchange.

control it's own blood flow.

Artery → Nutrient artery in tissue → Arteriole → terminal Art. → capp → venule. → veins.

capp → Pref. Chanels / True cappi.

Pref. Chanels → big cappil.

Arteriole →
Continous thick muscle fold. (dilation & contraction to increase or decrease blood flow).
Metar. → multiple folds but not continous.

Metart meeting capp. → pre cappillary Sphinchter.

Venule → can contract even if less muscle (muscle weak). because they have less pressure.

(*) Types of pores in capp:- 1) Inter cellular cleft → between Endo. cells.

(2) liver → featury of protein (protein size is large).
that's why the pores in liver much larger.

Kidney → in addition to cleft → fenestrae.
GIT → less than liver, more than Brain

Per vagina →
2 fingers.
Per rectum →
1 finger.

Median umbilical fold → Urachus
Medial 〃 〃 lig.
Letalteral Epigm.

1) Supravesical fossa → between median U.F & medial U.F
2) lateral, medial inguinal → between medial & lateral.

Pouches → accumulation of fluid during Infection.

In male only one pouch → Rectovesical pouch
Recto Uterine 〃 (douglas pouch).

Per rectum → If pro prostate have cancer.
douglas pouch → If filled with fluid means Infection

— x — x —

Greater Sac →

meso colon → pink in colour below Trans. colon.
anastomoses of (right) & (left) colon. Colic Art.

(Root of S.I)
from Jejunum oblique till Iliac fossa.

meso of Appendix → Appendicular Arteries, If blockage of Appen Art
Appendicitis → If rupture spread of infection. Engorgement

falciform lig. end → lig. teres.

lig. of Stomach → Gastro splenic, Gastro phrenic, Gastro colic, Gastro pancreatic.
left colic flexum. T. colon. Spleno renal or (lienorenal).

lesser sac → in back of great omentum, lesser omentum.
ant part or hilum of spleen + Ant. aspect of Kidney.

Epiploic foramen → no omentum.
or (omental foramen)
for reaching 3 structures portal vein ...

Right side

Folds & Recesses → Intersigmoid recess → V-shaped.
appendix → Retro cecal recess.

lesser omentum, descending of (perit. folds)
folds from peritoneum from diaph. → Liver,
attachment of Liver to diaphr. → hepato phrenic.
then from Liver to stomach — form lesser omentum in lesser curvature of stomach
(anterior & post portion)
from porta hepatis (till pylorus end part of stomach)

ligaments for more support → hepato gastric, hepato duodenal

very imp. Imp
3 structure.
Bile duct, portal vein, hepatic artery
I.V.C. lies

Greater omentum, 2 ant. 2 post.
4 layers (2 layers)
from G. curvature to transverse colon descend slightly downward & then reflect again to t. colon up to sup. part of pancreas
(surrounding transverse colon)

Greater Omentum → (storage of fat, but contain macrophages destroying of microorganisms)

Cavity between 3 & 2nd layer. (lesser sac)

Attachment of T. colon to post. wall
always ... Muse...

((Peritoneum))

Diaphram → lesser foramen (pelvic brim) → (Abdomen Cavity)
pelvic brim → floor → pelvic cavity.

Abdomin → peritoneum → parietal : → anter wall سس الداخل
→ visceral. (Invest) (Viscera or organs) وسه الخاك

peritoneum cavity → In male closed.
In female → open → peritoneum down completely cover the ovarier
ovarier has opening inside uterus & outside uterus
that's why peritoneum open in female.

Right Sub hepatic recess → accumulation of fluid.

but when standing → In pelvic part. due to gravity.

peritoneum covering whole surface of organ → Intra peri like S.I, stomach, jejunum, then upper part of duod
only 3 surfaces of organ → meso (ascending & descending colon). (liver, bladder upper part)
only anterior part " " → Retro (Kidney, bladder)
or (Inter peritoneum)

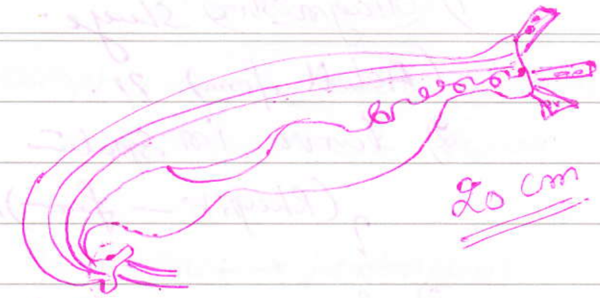
Prostate... → doesn't contain peritoneum

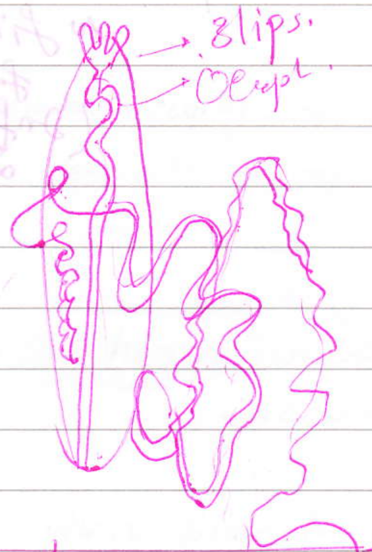
(How to aspirate Urine from Bladder without damaging the pleura.) perit on. ... ...

Peritoneum → fixing of Organs in position.

Omentu, stomach : parietal from Anterior & post.
, menentrie → S.I, meso → colon

1) diagnostic stage.
(Adult form) ??.
2) Larva in sputum
? (Rhapiti – form).
3) Rhap. → converted
to filiary
form
(Infective)
2.5 fold.



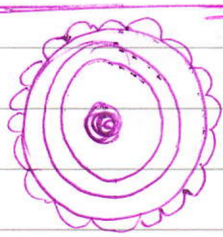


1) Blood vol.
2) R.B.C
3) Haematocrit
4) prod. of R.B.C
5) Stages of maturation of R.B.C
6) Factors regulating R.B.C prod.
7) Substance req. of production of R.B.C
8) Types of Anaemia..
9) Factors of R.B.C (Phtwo)
10) Formation of Hb
11) Storage & Transp.
12) destruction of R.B.C

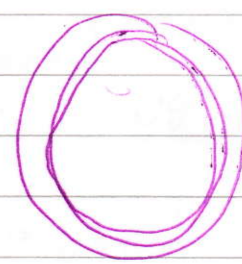
egg :- 1) Infective + diagnostic ??

4 pyrrole ⟶ protoporphyrin IX
prot. p. IX + Fe^{++} ⟶ heme
heme + polypept ⟶ hb α
2α + 2β HbA
O_2 10

2) Rhapidity from concentrated to filling for


proto blast


Blasto mere



1-7-0

⊛ Leishmania (Seminar) :-
—x—x—x—

Classification according to 1- Epidiomology. / G.D. / & Clinicaly.
due to DNA Technology
amastigote form :-

⊛ Mode of transmission :— ① contaminated Isoenzyme needle pricks. (rarely)

flagela → flagillin → which highly initiate immunoresponse
↓
Retraction of flagella before entering.

② Cover itself by membrane to protect from lysosymal enzyme.

→ ((Spleen may cause pain in late stages))

{ Cell mediated → T cells + macrophages (assist).
{ Humoral → (Antibodies) Antigen reaction
↓
لازم يشتغلوا مع بعض
ولو حد فيهم اشتغل لوحده
Ig فازدياد
destruction of macrophages

} In children → sometimes (local Anasthesia)

Neomonikl.

transp. Glut 3 independ.

Triglyceride storage occurs in muscles.
(glucose is primary source of Energy).
Brain → only 2% of Total body weight.

[illegible] RBC — 140 gm of glucose required by brain daily.

· Why Brain & Liver are both insulin independ. but both have diff. Glut 2,3 ??

because by consumption of glucose by both, glucose level decreases, thus glu2 when low level prevent going to (liver) → another source but brain requires energy small of energy but continuously & doesn't have another source of Energy only Ketone if in high level in severe Starvation.

that's why Glut (3) → absorb or transport glucose even if in small amount. (due to Brain Blood barrier.)

Starvation :-

glucose ↓, a.a ↓, all are low, degradation ↑.

1) Avail. of Substr. ↓. (degrad).
2) Allost → F 2,6 Biphos. is not availability due to low Insulin, gluconeo is not inhibit enzymes are available by phosp.
3) HM - Reduction
4) de novo synth, Citrate NADPH all are not available.

Adipose tissue :- → adipocytes could be seen by naked eye due to large size

70 kg → total body weight

(adipose half of total muscle mass) → 14 kg.

Skeletal muscle → Aerobic & non aerobic glyco.

↓

rest پر کم ہو
اور زیادہ
& excessive

But in rest only 30% of O_2 consumed by body through muscles at rest.

But in heavy excersize → 90%.

Brain → 20% during rest due to presence of Growth hormone during sleeping & resting more active.

---

diff. between cardiac & skeletal :-

Cardiac	Skeletal
1 non volun.	1. Voluntary.
2- aerobic	2- aerobic & non aerobic
3- no storage of glycogen	3- Storage of glycogen

Glucose uptake by SK. m. → Insulin dep.

glycolysis in S.M → for energy mainly.

protein synthesis → ↑ 3 branched a.a is used by S.M in protein synth & Energy production

↓

high, cause high amount of protein is lost

CHO ~~ Acetyl CoA → Cholest
CHO ↙ Acetyl CoA → de novo
CHO ← AcetylCoA → Keton bodies
from fatty acid.

The main tissue of denovo fatty acid synth → liver.
Adipose → only storage.

Liver is the main site for synth :- "Availability of substrat Acetyl CoA in liver is high. 60 gm from every 100 gm of glucose goes to live.

(2) availability of. NADPH.

(3) Citrate high in amount due to high amount of glucose & citrate gives high amount of Acetyl CoA.

Citrate → activator of Acetyl CoA carbox. (denovo)
Citrate degra → Acetyl CoA → B- Oxidat.

Allosteric activator. high amount of fat in liver → expell it out by circulation in form of VLDL (Triglycer). Store in adipose.
If stored in liver → fatty liver.

Chylo. remnant → degred → release of fatty acids.
cholest. esterase.

Liver can metabolise → all a.a except Isoleuci- Leucin, valine → branched a.a no enzyme.

⊕ feedback → 1) death.
2) pregnancy → more contraction more effects.

Baroreceptors. / Afferent → Vagus, glossopharyngeal.
↑ Stimulant → pressure stretching

كلما زادت الضغط كلما زاد عدد الإشارات الكهربائية.

↓ Responce → neg. feedback, vaso dilation, B.P ↓

Baroreceptor → (decrease & increase pressure)
↓ reset due to stable pressure
(Postural)

Symp. nerve fiber in kidney → improve setting.

chemoreceptor → like barro but diff. in nature of receptor.
Stimulation of vaso con ys
Inhibition of para

low. pressure recept → in places where pressure is low.
Atria → O.
Pul. v → O.
nature → (Stretch), venous return
Blood vol. ↑ → Atrium → Stretch of wall.
Brain medulla
Vol ↑
H.R ↑
cont ↑

diff. between higher brain centers & vasomotor :-

controls if the action can occur or not
responsible for consicousness. eg :- (Urination)

works automatically without consciousness.

(التبول مثلاً).

Vasomotor tone → without any stimulation, only by sympathetic discharge.

Action on heart & vessels → (together)

Epinephrin → Vasodilation in skeletal mus & some coronary Art.

تعتبر vaso dilat في بعض الاوعية

Excersice → في العضلات
(Vasodilation by local control)

Emotional fainting → hypo tension, Blood flow ↓, vaso dilation

In muscles 2 effects → 1) local mechanism. Vaso dilation of muscle tissue
2) Stimulation of Vaso constrictor area → vaso dilated muscle.
Increase B. flow

Symp. vaso dilation doesn't occur because only at beg. of Excersize.

(not localy but globaly).

Nervous syst. → not only local control, but more global function.

Redistribution → not in indivisial vessels locally but as segments

B.P control :- 1) Nervous → rapid. 2) Hormonal → long. 3 Kidney

Brain Stem :- 1) ~~Cortex~~ mid brain 2) pons 3) medulla. } vasomotor.

⊛ Cappil, met; precap. sphi- → responsible for local control that's why.

(constriction of Arterial → not Innervated. large displacement of Blood towards the cappillaries) because near the tissue.

Skin → due to regulation of Temp., (Thermoregy), Cold :- constriction. hot :- dilation.

<u>Comp. of Reflex:-</u> Sensory receptors / stimulation. → Afferent fibers. → Center vaso motor. (Brain, Spinal cord) مصدر الأوامر → efferent fibers → Organs (Effector) → <u>response.</u>

(3) CNS ischemic Response :-
Stimulator :- mainly accumulated CO_2.
Action :- direct stimulation of vaso consti. & cardio accel.
high Increase in B.P.

→ may cause occlusion in some peripheral vessels such as in kidney.

(Cushing Response) → Imp. in reducing Pres of C.S.F when reaches more than B.P.

(4) Atrial & pulm. reflex. → low pressure receptors.
Stimulant → change in vol.
Action :- → Kidney :- Afferent arteriole dilation. more filtration.
hypothalam :- decrease secretion of ADH.
Combined action → Increase urination & decrease B.P.

(5) Volume reflex :-
(6) Bain Bridge r :- Increase in H.R 75% → 15% by stretching → remaining by stimulation of vaso cont. region by receptors that cause nervous Bain Bridge reflex.

(*) Nervour global control:-

Neg. feed back mechanisms of Ner. control of B.P:- (reflex)

① Baroreceptor control (Baroreflex).

Stimulator :- mainly high blood pr. also low B.P but more than 50-60mmHg

Action:- by strech receptors.

(decrease the Artrial pressure by ~~stim~~ Inhibition of Vaso const. region & cardiacceleration region) & Stimulation of para Symp. Inhibitory region.

also causes Increase B.P.

Imp:- 1) The most imp. reflex mech. rapid.

2) Considered as Buffer reflex.

3) Most imp. in preventing minute to minute change in B.P during change in posture.

② Chemo receptor :-

Stimulator :- low O_2, $CO_2\uparrow$, $H^+\uparrow$.

Action :- Stimulation of Vaso constric. & cardio accele. & thus (Increasing) B.P.

→ But only when B.P is lower than 80mm Hg.

يبقى الضغط عالي بسبب وجود ارتفاع PR
مش بيزيد أوي في

diff between Volume loading H.T & Essential H.T in course

V.L.H.T → not PR↑ but cardiac output↑.

E.H.T → due to mainly Increase in P.R.

Ang. II → Indirect retain of Na + water

Renin - Angi system → Increase & decrease pressure in both cases.

during shock → first mechanism (Nervous control.)

then renin ang. Vasoconstr. then Long term

بيبدأ أولاً «رسمس» ويبقى الضغط بيتغير قليل.

(*) Affect of Increased Ang. II → imp. (direct & Indirect).

(*) Feed back mechanism:-

(Converting enzyme Inhibitor) → drug.

في حالة P.R ← لم يتم أي زيادة في Vol. مع ذلك تعمل Kid. وتعمل على إخراج كميات كبيرة من البول لكي تخفف الضغط
زيادة مؤقتة في A.P نتيجة عمل الكلى Renal Body fluid syst.

Diebeties & chronic hypertension → Increase vessel resistence inside kidney.

B.V → part of E.C.F

Auto regulation → Increase in B.P causes ↑ Blood flow

B.P↑ ← Increase in P.R ↔ Vasoconstriction of vessels in body by tissue. (metaarteriole precap. sphincters)

—x— x — x —

Salt-sensitive hypertension → affected by NaCl.

Salt non-sensitive " → no affect of NaCl

Normal range of pressure → the pressure which doesn't cause any damage to tissues & kidney, even minimal.

Heart → Over load of pumping due to increase press
↳ (damage)

Retina → capillary, Arteriole, veins → stenosis.

Brain → rupture of vessels, cerebral stenosis (stroke)
damage

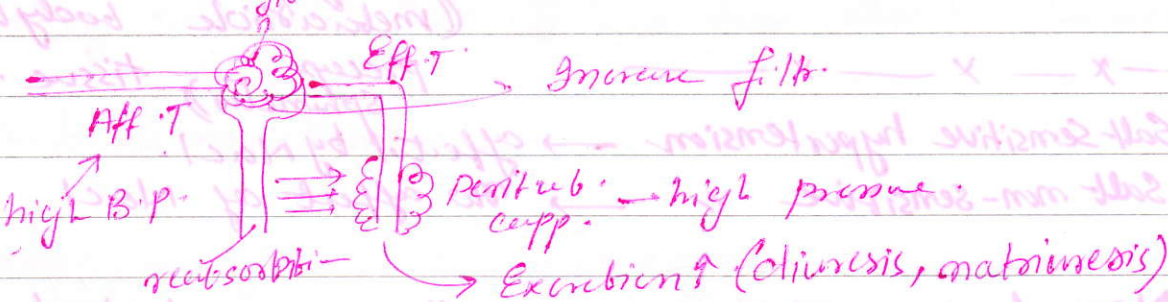
Kidney → stenosis of vessels inside → Renal failure.

⊛ Kidney Regulation :- 1) Rapid → Sec → hours.
—×—×—×—
2) Int. → min → days.
3) Long.

→ Renal Body fluid mechan. → imp. that kidney function should be normal
depend on :- pr. natriuresis.
pr. diuresis.

Vaso/cons

→ when there's increase in blood vol. → increase in Blood pressure in Afferent Arteriole → ↑ filtration in renal glomeruli → ↑ pressure in peritubular cappillaries as a result water & salt can't be reabsorbed by tubules & is excreted in large amount in urine → B.P↓.



ADH → Secreated in low pressure state.

كلما زاد pressure curve يزداد Out put أكثر من Input ← لين يرجع الوضع إلى Equillibrium

. retention of Salt & water وكذا Out put يقل pressure curve كلما
ADH, ←

1) dehydration 2) Diarrhea 3) Heamorage.

Kidney act on volume to maintain pressure.

(Integre Multifa syst)

1) How pres. control mech. can be sorted according to their rapidity in action.

2) What are mech. & what are their fixed action.

3) " " Intermediate control mech. & how each of them work.

4) For how long effect of Int. mech. last & what's benifit of this. → 30 min to several hours.

5) Summarize the Interaction of long Term mech by Kidney.

→ Which of the comp. of this system has powful constrictor effector:

→ What are two principal effect of Ang. II that leads to Increase Pressure.

→ Use 19-10 to discuss the rapidity & intensity of Vasoc response of reni.– ang. II.

→ What's the effect R-A sys on body salt & water.

→ Whats indirect mechans. of renal retention. of salt (Aldosterone)

→ " the role of R.A sys in maintaing Arterial pressure when salt intake increase.

→ (Hypertension caused by R-I dist)

→ What's principal cause of H.T involving R-I system.

→ What are renal conditi– that might cause increase renal secr & H.T.

1) Tumor

2) Continuous Infusion of Angiotensi–

→ (Essential H.T).

1) what's ↑

2) What are main charac. of E.H.T.

3) " " physiological base of Treatment.

(*) Kidney reg.

1) ~~Dominant role of kidney~~

1) Renal body - f syst for arter

→ System is primitive one.
→ Pressure diuresis and net.
→ discuss fig 19-3 how pressure return to E. point by this system.
→ Why increase in T.P.R fail to elevate long term
→ how ~~[illegible]~~ Increase fluid fluid intake Elevate press if & Renal normal.
→ What the imp. of salt.
→ define hypertension, what's the vol. loading type of H.T.
→ List changes in circulation
→ (Role of Renal Angio.
→ What's the role of R.A.
→ Where (Renin) come from.
→ How Renin is converted to Angiotensin II.

⊛ **Notes:-** Changes in Circulation as result of developing Volume-loading Hypertension:-

1) The part of kidney has been removed, leaving only 30% of normal mass.
2) high amount of salt solution given, which lead to increase in E.C.F & Blood.V & Cardiac output as a result of impairment of kidney to release the excess fluid.
3) Result in Increase Arterial Blood pres. ⟶
4) Peripheral Resistance ↓, due to action of Baroreceptors. C.O, E.C.F, B.V↑.
5) After some days P.R ↑, due to adaptation (reset) of Baroreceptors to new pressure.
6) C.O, E.C.F, B.V ↓ ... as an Effect of Autoregulation

Note:- AutoRegulation ⟶ decreases, CO but Incr. P.R.

7) As a result Increased Arterial tension ⟶ P.R↑.

Volume loading hy.T. :- 1) Increase in B.P due to ↑ E.C.F, B.V, C.O. (Primary)
2) Increase in B.P due to P.R ↑, even if C.O↓. (Secondary)

Note:- P.R doesn't cause Increase B.P but it's Secondary to B.P ↑.

Total changes of circulation :- 1) Hypertension. 2) Increase P.R.
3) decrease of C.O, E.C.F & B.V to normal values.

decrease in C.O → 1) Autoregulation.

2) Vaso constriction as a result of ~~decrease~~ Incr. P.R and decrease the cappillary pressure, fluid accumulated between tissues absorbed into the blood by cappillaries (pressure ↑ → pressure ↓).

3) Increase B.V is excreated through the kidney.

⊛ Types of Volume Loading hypertension:-

1) Decreased Renal mass & Simultaneous Increase uptake of NaCl.

2) Excess Aldosterone in the body.

pri. Ald.

When glycolysis ↑ → gluco neogenis ↓
because pyruvate → (Acetyl) coA (high amount).
↓
Oxalo Ac cycle OAA.

no relation of acetyl coA obtained from glycolysis in activation of gluconeogenisis.

but gluconeogenis is activated by high amount of Acetyl coA which results from lipolysis & then activates enzyme phosph carboxylase key enzyme of gluconeogenis.

— x — x — x — x —

Insulin decrease the level of glucose by increasing it's ~~absorbti~~ uptake by tissues.

5 transpor of glucose

Insulin depended tissues some of them not depends only requ transporters such as (Glu 2, 3, 4)

glut (2) → In (liver) → insullin independent.

well fed → Increase in glucose phosphorylation.
due to activation of glucokinase when glucose 6-p level increase inhibibit (Hexokinase) → لا يثبط الانزيم / علاقة زيادة الجلوكوز Km↑

glycogenisis ↑ → glycogen synthase activated by ↑availabity of (G-6-P) → substrait & activator of enzyme.
2) dephosp. by Insulin → activation.

HMP↑ cholesrol↑ lipogenisis↑.
NADPH → synthesis & defence (glutathione reductase).
2) Aldose reductase → of eye NADPH.
3) also during protection against Invasion

HMP CoA reductase NADPH dependent.

(HMP) → consumes 5-10% of glucose metabolised in liver 60%.

glycolysis ↑ → due to activity of phosphofructo I & pyruvate dhase.

not as glycolysis enzyme, but as joining between glycolysis & kreb's cycle

The most imp. allosteric activator of glycolysis & inhibitor of gluconeogenesis
(Fructose 2,6 Biphosphate)

① activator of key enzyme of glyco → phosph...

another example of Allosteric enzyme activity :-

② Cytrate key enzyme of lipogenesis is activated

Acetyl CoA

Induction & repression → Insulin goes to gene & increase the transcription

eg. of enzymes (Increase in no. of active site).

1) phosphofructo kinase ↓ or decrease

2) Pyruvate dh complex.

3) HMG reductase of cholesterol.

but in allosteric no increase

Main organ in metabolism → (Liver) because all metabolites by portal vein goes to Liver.

glucose, chylom. remnant etc.

لأنه المكان الحرج circulation.

Liver :- produces more than consumes, every 100 gm of glucose entering 60% taken by Liver & the ... from this most of it storage the remaining energy.

In Allosteric activation of covalent modification → no increase in no. of enzyme only activation

but in Induction & repression → Increase in no. of active sites

* Induction → تؤدي الى زيادة التصنيع
وليس الى زيادة النشاط الإنزيمي
no activation of enzyme.
كمية الإنزيم فقط تزداد.

glucogon & Epinephrin → * phosphorylation or dephos.
active to → inactive or
inactive to → active.

Insulin → dephosphorylation.

* all the enzymes in your body is active in present of Insulin & dephosph. except 3 enzymes:-
*(Glycogen phosphorylase & glyco.phos. kinase.
glycogenolysis
hormone sensitive lipase) → active if phosphorylated.
lipolysis.

Phosphatase → dephosphorylation.
all body in well-feed state will depend to obtain energy from glucose.
أدوات التحكم في المواد يستخدم طريقة كل شئ
4 mechanisms regulating → ① availability of substrate
② Allosteric
③ Covalent modifi.
④ Induction & repression
تحدد أي منهم

((Feed / Fast cycle))

digestion & then absorption → balance should be between diff. types of food, carbohyd., fats, protein

Absorption time → ~~[illegible]~~ 2-4 absorptive state.

After absorption increase of glucose & a.a in blood & triglyceride in form of chylomicrone only.

according to type of food. if normal but if more fats till to 6 hours.

Chylomicrone will ~~[illegible]~~ reach liver in form of chylomicrone remenents only

liver only receptors for chylomicrone remenent only.

Chylomicron goes to → Adipose tissue, Skeletal, & cardiac muscle

lipoprotein lipase → found in these tissues convert triglyceride into free faty acid + glycerol

free faty acid → adipose

glycerol → liver due to enzyme glycerol kinase

Ig ratio :- $\frac{I}{g}$ high ration

Insulin / glucagon

Insulin → Storage role. (Synthesis) → glycolysis → Energy & fat formation.

while in case of protein → بتسابق علي البروتين

a.a وحدة البروتين والكلة لا تخزن يتم تكسيرة

~~[illegible]~~ nitrogen compounds → protein

protein non nitrogenic → neuclein acid

Insulin → Synthesis of all ↑

Induction of key enzyme

SABIN-
FELDMAN

living Antigens of Toxoplasma
adding Virunale to Mg + Ca found in blood
+ methylene blue
complement will be activated

preparation of Serum in diff. dilutions.
from each one prepare Giemsa stain after each 7 days then we will see which in dilution the toxoplasma is disappeared more than 50%. (unstained) → positive. disappearance of cells.

(highest dilution with normal saline of patient with chose positive serum Antigen-Antibody reaction)
→ Titration.

⊕ Adding of Antibodies from patient to toxoplasm of laboratory at 1/800 highest titer at which posit. anti-antibody reaction occurs
+
The rule of 3 Twos. if 1/800 after 2 weeks → 1/3200 (Active Infection)) but if chronic - no change.

Serological :- ① highly dangerous specimen in laboratory.
② the specimen : Serum & blood.
③ For any Serological test. See the control.
④ Always use rule of 3 twos in titration)) method.

→ effect eyes in high percentage.

* Toxoplasmosis :- ① Chorioretenitis in 99% of cases, and Bilateral chorioretinitis in 85% of cases.

② Hydrocephalus or Microcephalus in 50% of cases.

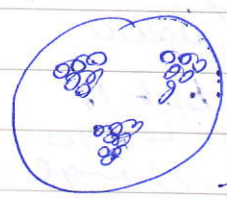
③ Mental Retardations

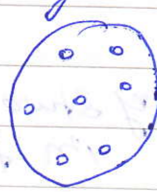
Algerian Sheeps → natural Immunity to Inbucks.
Cows → " " " Toxoplasmoses (Asymptomatic)

but the rest of animals → can develop complications.

Clinical picture among immunocoporent. page (18), a, b, c, d
(H.W)

Every week the cells are transported from culture to culture from tube to tube & new Hank's solution is added to keep it alive → transmitted from the infected women
→ any virus can eat and (Hela - cells) → woman's name from cervix.
leave remenents by seeing the nature of these remenents we can distinguish the type of virus.


→ polyomyletis.


→ Influenza virus

we keep these cells reserved in order to know the type of virus

(Frothy stool → due to malabsorption of carbohydrates).

Thick layer → pars distalis.

Epith → low columnar Intermedia.

acidophilic → orange, pink

base → dark blue.

Chromophobe → very pale, acidophilic → pale but darker

Basophils → densest.

→ cords  outside cappillaries.

neurohypophysis → Alum Heamtoxylin → Herring bodies.

triangular cells → pitucytes.

(ill defined septa → of thyroid).

Parathyroid → Straight sheets or clumps of cells.

Thyroid cells → contains small vacules known as (Scaloops)

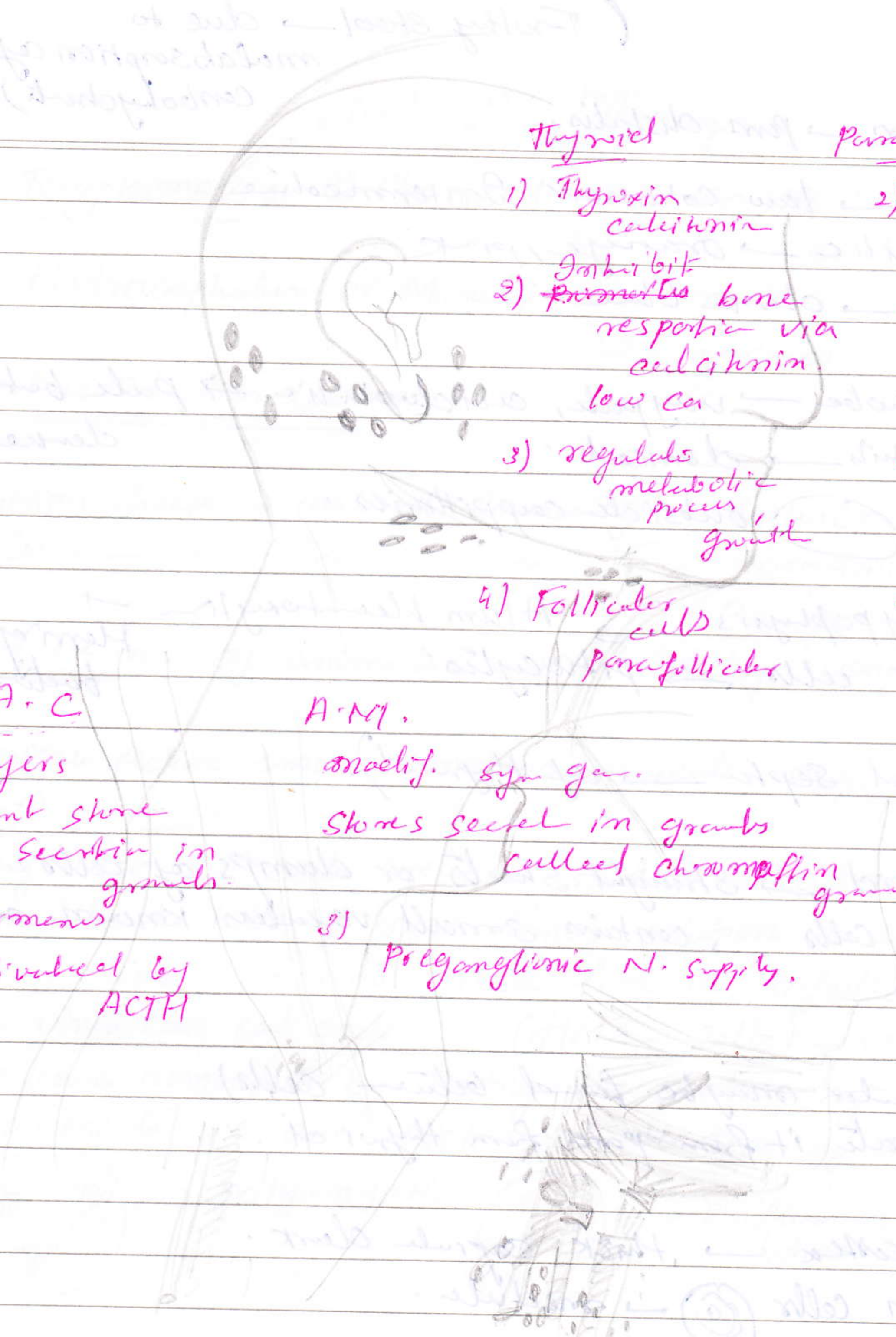
(parafollicular may be found between cells)

C.T seperate it from para from thyroid.

Adrenal cortex → thick capsule dark.

ganglion cells  → medulla.

Brown granules in cytos → Chromaffin.



Thyroid	Parathyroid
1) Thyroxin calcitonin	2). Parathyroid hormone
2) Inhibit ~~[illegible]~~ bone resorption via calcitonin low Ca	3) Ca ↑ intestine resorption
3) regulate metabolic process growth	maintain of bala. of Ca p.
4) Follicular cells Parafollicular	cheif cells oxyphil cells

A.C
1) 3 layers
2) doesnt store secretion in granules
3) hormones
4) Activated by ACTH

A.M.
modif. sym gan.
Stores secret in granules called chromaffin granule.
3) Preganglionic N. supply.

⊛ Malaria (2) - Seminar :-

New vaccine against malaria by Galaxo :-

(R T S, S → antigen from hepatocyte.)

R → Antigen, T → T cell, S, S → segment (from yeast antigen)

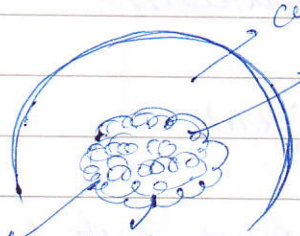
→ diagnosis nowadays not on clinical picture but by taking samples of blood.

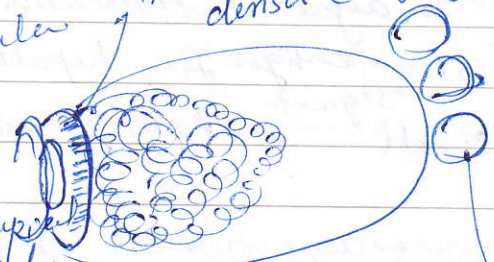
R.B.C in memb. has functional prot. if impairment of prot. such as glycophorin which results in Duffy grp. But in normal cond. (glycoph) present in general in memb. of R.B.C receptor.

Spleen → destruction of HbS but compensation by HbA.

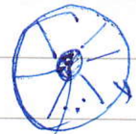
(Adenohyp.)	(Neurohypo)
1) 3 parts	3 parts.
2) Blood supply, capillaries no nerve supply.	nerve supply, axons, neurons. glial cells.
3) Oral Ectoderm (Rathke's pouch)	Neural tissue.
4) 2 types of cells secrete many types of hormones production	only secretion but not product of vaso, Oxy..
5) hypothal.-hypoglossal portal system mainly blood supply by secondary capillary plexus	Inferior hypophysis artery & capillary plexus.

Cortex:- Renal Corpascals.

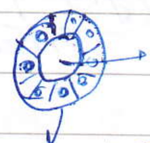




P.C.T.



D.C.T.



1) Sterile lanset 2) thick film / thin film by Leishman Stainer
3 drops 1 drop. Geimsa.

for malarya parasite.

Seminar histology:- Cortex → dark area.
due to presence of Renal corpuscal.

Parenchyma → cortex, medula.
cortical -

medula → lighter area.

lumen small  → P.C.T.
" large → D.C.T.
Bladder → no submucosa.

⊕ Cestodes:-

→ Hydatid - Disease

→ " cyst . caused by Echinococcus {granuloses}

rupture of cyst → Toxic

Taking all the cyst out to prevent recurrent development of cyst.

الدودة الشريطية الكلبية

Specimen → Bone marrow Aspiration. , in association with, Hb, WBC count, platelet count

non specific result could be positive in any other disease. Albumin → less. Gama globulin → raise.

highly specific, highly sensitive test → Tibia, p. Iliac, A. Iliac Aspiration.

→ promastigote form of Leishmania donovani & Infantum.

→ ~~protozoa of under phase contrast.~~ ~~promastigote form~~

→ Naglaria flori & Acantamoeba → amoebic of C.N.S. meningo ulceration ↘ very rare but fatal in few days.

→ Substances can seen in phase contrast microscope. ← imp.

Cerebral spinal fluid seen in specimen. phase contrast microscope because organ highly transparent

In light microscope → no picture highly transparent.

Blood & Urine, C.S.F → purely sterile in normal condition but presence of moving particals means one of 3 particles.

Jejenum → high intestinal gland in lamina propria.



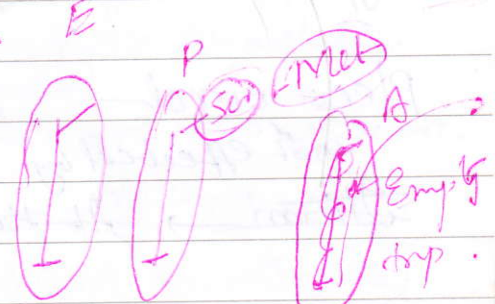
deudenum → high intestinal glands in submucosa

Ilium → Scattered villi

Hexyl's layer founds in (hair follicle)

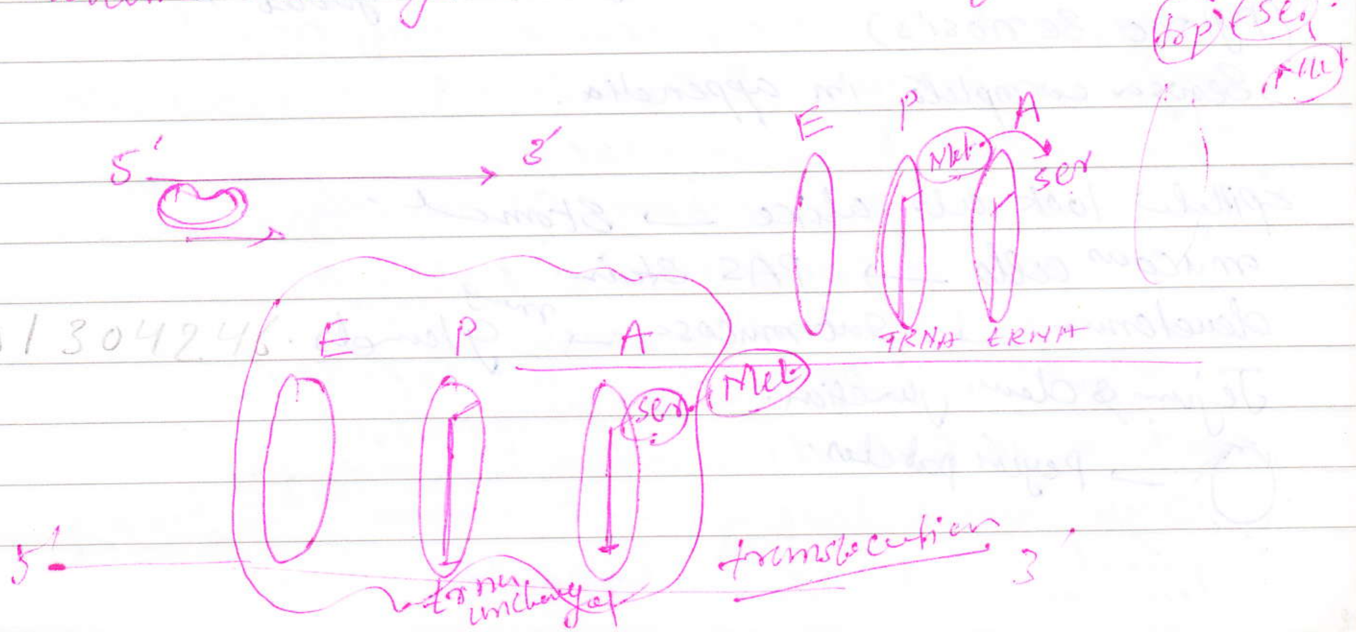
Paciniar corpuscle → deep pressure recep—.





Optic nerve when pierce Sclera → become mylenated.

Muller → Neuro glial cells.



Paneth's → acid cell.
→ contains Zinc.
↳ Orange A) Impre.

(Stomach)
mucosa → rugae / pits.
all cells looks alike.
Argentaffin (Entero Endo) → due to Silver impreg.
foamy cyto. no goblet cells
Serous acini like → no brush.
zymogenic cells.
secretion of Serotonin (Enterchrom.

Submucosa → very bulky after —
Conducting portion is 2/3, Secretory 1/3 → (Pylorus mucous acini tortous).

Crypts → duod, jej., ilium → & appendix.

Plica circ. → evagination of Submucosa
not effected by distention covered by mucosa.
~~Ilium~~ → ~~At the~~ they are permanent
they disappear at the mid of Ilium.
8 mm in jej.

3 types of cells in large int.
Columnar, argentophil, goblet.

(Pyloric Senosis)
Serosa complete in appendix.

Epithel look all alike → Stomach.
mucous cells → PAS Stain.
duodenum → submucosa → many glands.
Jejum & ileum junction.
 → Peyer's patches.


→ Secondary C.T. papillae: Inside the papilla.

Tongue → highly Elevated papillae
'filli' form --- . we can see it very clearly.

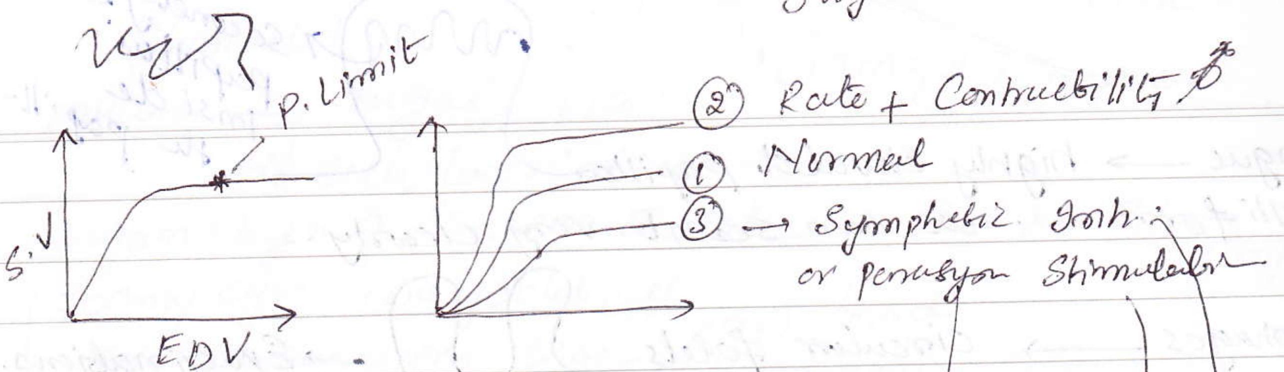
Esophagos → circular folds → Evaginations.

lip → External Surface is thin, while Internal thicker
contain Stratified squ. Epith, below it
we can't see ~~mucosa~~ musculamis mucosa or other

@ parasitology :- Octamitidae → 4 pairs 8 flagellates
previously was known as hexa → Six flagellates.
Giardia (Aglis, moris, Intestinalis) → length more than wideness.
mice &
→ Only protozoa for human
Trichomonadidae → 3 flagellae

Aglis →


Inogen Stimul →.



Actin/myosin → after p. Limit, tension increases which causes.

doesn't contr. force. | ~~Rooms~~ | due to presence of Time. 30%.

— x — x — x —

(Histology Seminar)

function of Stratified. , Liver, pancreas → (glands outside the organ)

glands → lamina prop.
↘ submucosa

Carmine Stain → demonstrating myosigine in mucous.

circular → peristalitis

longit → segmented movement.

high folds of Esophagus → when Esophagus is empty.

during food swallowing become longitudinal.

Ridges → for stress. , Oral Cavity → lips (mucosa)

Internal aspect → very thick.

Tongue → many directions of muscles. — no submucosa in tongue.

→ partially Keratinized, post asp. →

ventral aspect → wet Epithelium.
↓ lower
fillifom → no papillae roughness

Cavity in taste buds → (polysaccarides) (Saliva)

Circumvallete base

peripheral mucosa. · (Serous) acini)

pressure in ventric. should increase more than that found in Aorta to open the semilunar valve.

after big contraction ← Slow سبب في more than 120.
due to the presence of Ejection diastole لي

Plateau the ventricle remains contracted.

(No Contraction)

لأنه لازم الضغط في ال Vent يبدأ أعلى بكثير من في Aorta.

Isovolumic — Closing of values passively

العضلة الزائدة. Pappillary muscle → prevent the opening (bulging) of valve into Atrium due to presence of high pressure in Ventri (Reumatic fever). during vent. contraction.

وظيفتها تمنع الصمام من الإرتداد للخلف.

EDV → amount of blood in (not filling) vent., at beg. of cont. & beg of systol & Ending diastole.
مو حجم.

not at the end of Ejection → E-S-V.
at " " systol.

Cardiac output → Stroke Vol. × H.R. /min.
Sympathetic only Increases S.V.
But F-St. → Relation of EDV to S.V.

① ED.V ② muscle length ③ Contraction
or Venous Return.
& Rt. atrial pre. muscle length)
3 factors of S. Frank

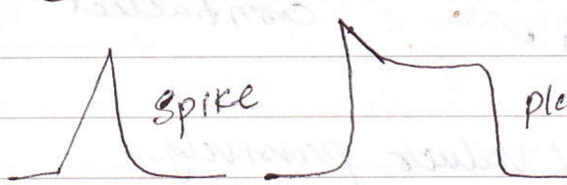
Seminar (1)

- Structural diff between Cardiac & Skeletal muscles.

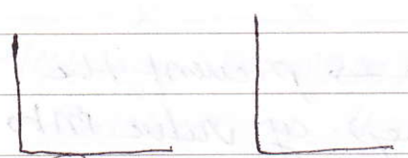
function of (1) gap junctions → rapid trans. of A.P for rapid contraction.
I.C.D.

(2) major source of Ca^{++}. Skelet → S.R
Cardiac → E.C.F or T-tubule.
Because cardiac muscle work as synsitium.

(3) T-tubule structure in cardiac muscle → more & larger.
→ Stimulation of Sarcopla. by T-tubule.



S.M → Fast sodium & K channels.
C.M → Slow K & Ca channels



(4) (A.P)
The both Ca & Na fast channels opens at the same time at rising phase of dep., but Na fast first because it's fast, at spike the slow ch remain opened.
(plateau continuation of dep.)
Events → only opening, of channels & closing.
of dep./rep.

(5) Stimulation of S.M → neurogenic, cardiac → myogenic.
from self excit. from S.A node.
Sym./para. only incre. & decrease for adaptation of heart but not due to Essential.

(Cardiac cycl → mechanical Event)

(Diastolic phase of ventricals) 3 phases (A.V open)
[illegible] P.G [illegible]
Atri. opening of A.V → P.G / amount of blood in Atrium.
phase (2) → cont.
P.G → decreases.
So slow flowing of blood.

Cyst in carriers may cause abdominal pain → due to feeding on bacteria
(*) (FEC method — coz cyst maybe little in carriers that's why this concentration method is used.)

Target organ → Liver, Intestine, brain, lung.
Type of lesion → Ulcers of Intestine due to proteolytic enzymes.

Bloody diarrea.
↳ mucosa, submucosa, muscles, Blood cappil. → Liver or Brain.
(layers of Invasion)

(E.h goes & flows in Veins, not in arteries)

480 millions infected all over the world but only 10% Acute the remaining either carriers or Chronic.
↓ fed on R.B.C. — Chronic → not fed on R.B.C

Cyst form are found outside the body, after entering may be converted to trophozoid (Acute cases) or remain as cyst (Chronic case). may be released in stool as (cyst) or trophozoid. but dead.
← diagnosed for stage & Infective stage.

Forma minuta → Avirulent
Forma magna → R.B.Cs, ...

Cyst form if is found in children should be treated immedi- cause due to low Immunity may be converted to Acute dysentry.
(periodic Examination) → fir

Giardia → very bad Odor of stool.

③ Classification of E.h :— ① Domain → Eucaryotes.
② Kingdom → protista.
③ Sub King. → protozoa.
④ ~~Phylum~~ Genus → Entamoeba
⑤ Species → ⑦...

✓⑥ E. gingivalis → maybe opportunistic parasite in teeth.

✓⑦ Acanthamoeba → Ulceration of Cornea due to lenses.

✓⑧ Naglerea species. Flori. → (meningoencephalitis) through Olefactory nerves causing brain abcess in contaminated water pools.

④ Morphology :— "cyst"

"Trophozoid" (Vegetative form)

diff. between Coli & h. → The Vacouls R.B.C, yeast.

main E.h → 5 ~~µm~~ / ~~hours~~ sec. (One direction)
Coli → Sluggish. ↘ (In fresh specimen)

Pathogenesis → ① Port of Entry. (feaco oral) Oral cavity ② Habitat ③ Attributes of Organism (Virulance, high). predespostic factors. Sometimes the Life style of human. (Unhygienic conditions)
④ Targent Organ or tissue.
⑤ Type of Lession.

2/10/013 - Wed.

② Parrasitology:- ① Introduction
② E.h → Classification.
→ morphology & Life cycle.
→ pathogenisis.
→ Lab diagnosis.

Parrasites (Triangle)

Host — Vectors → Sometimes present & Sometime not.

Facultative → Sometimes Opportunistic.
Obligatory → malaria, E.h., T.V.

definitive host → (human host).
well mature form, Sexual, adult form.
(Trophozoid cyst form)

Intermediate → Sometimes parasite require 2 hosts primary / secondary.

Reservoir → preserve the parasites in nature.

Vectors → always are Insects.

mechanical (carrying the parasite only through insects)

(Biological) & definitive host.
→ Parasite found inside the Insect itself like Anophilis mosqitoes).

1) Genes 2) Species 3) Strain.
→ high Virulent,
Law ".

→ Entameaba histolytica highly Virulent
(genes) (Species) (Strain).

AIDS → Avirulent or highly virulent according
(HIV) to the condition of the patients.
but if immunity lowers it becomes Virulense.

eg. Entameaba histolytica → normaly only diarrehea.
but may lead to destruction
of liver.

⊕ Subcutanous tissue parasites:-

—x—x—x—x—

⊛ "Entameaba Histolytica":- feaca-oral transmission — 19/9/013 Thursday

- liver absess,

Stool
Test:- taking a part of stool & mixing with normal saline Nacl and cover slide → Unstained only chromidial bodies visible no nucleus. But if we add a drop of Iodine (Logul's solution) we are diff. E. coli from hystolitica nucleus will appear & Chromoses bar disappear; becaus E. coli has 8 nuclei, while E.H 4 nuclei. & E. coli nuclei appear in stained & unstained.

→ for every parasite Infective Stage:-
fully developed 4 nuclei, cyst without chromatoid

No vaccination for parasites, → malaria, ∴ coz the immune System response is very weak & poor but for bacteria, Virus Specific.

⊛ Microbiology (Clinical Microbiology).
① C. parasitology ② C. Bacteriology ③ C. Mycology.
④ C. Virology ⑤ C. Immuno sero logy.

3 main bacterias found in milk.
⊛ food - Microbiology :- 1) Salmonella Typhy, 2) Brucella, 3) microbacterium.
⊛ Sanitaro epidemiology microbiology. الوبائية → To detect desease in carriers, taking Specimens from diff. ages, & giving precautions
⊛ Industrial microbiology.
⊛ Cosmos Microbiology.
⊛ Environmental " "
⊛ Sea & Oceans "

food microbiology → To detect the parasites & precaution.

If an examination is applied on 100 young white mice and a parasite has infected even only one of them, then this parasite is pathogenic to the human either :-
high Virulence, Medium Virulent, Low Virulent, or Avirulent But pathogenic
(But Comunal are not Avirulant because they are pathogenic)
(Comensal is non-pathogenic)

17/9/013 / Tuesday...

Medical parasitology
Introduction

Genes name → Starts with Capital letter :-
eg:- Entamoeba ~~histolytica~~
Species always starts with small letter. eg. histolytica.
Plasmodium falciparum. → Nomenclature.
(Genes) (Species)

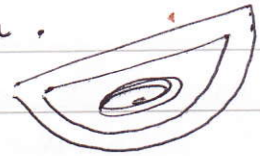
phylium, Family, Genes, Species.
(الشعبة) → الموطن الأصلي (in which country, & distribution)
(Habitats) (Geographical location? of parasite inside the body.
plasmodia falciparum → destroy R.B.C (habitat).

Morphology :- Active form, cyst form, Egg of parasites.
(drawings & Explanation). fertilized, unfertilized.
→ Cyst-form
→ Trophozoite (active) form.
→ Eggs.
protozoa don't have separate male or female but instead (cyst) in gerdia, ameaba. we write only (cyst or Trophozoite) but not egg. because female can't form egg.
(But in Nematodes, Cestodes, Trematodes) → we can say eggs.

* Protozoa :- 1) E.h 2) Giardea lamblia 3) Trichomones vaginalis 4) Toxoplasma gondii 5) Malaria parasite.

Egg of Ascaris lumbricoides contains fertilized segmented Ovum.

* Egg of Enterobias Vermicil. contains Active, mobile larva.



* Pathology:- eg. R.B.C is destroyed & B.V is blocked by plasmodium falciparum & it can abact any organ.

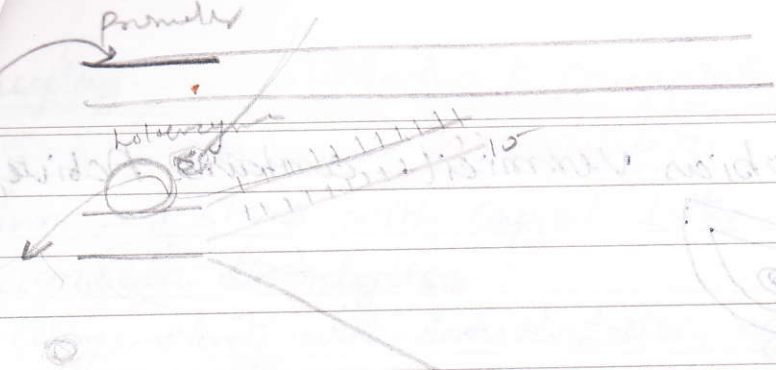
* clinical picture & (pathology) is different.
↓ (Action of malaria parasites into the body.)
Symptoms.
الأعراض * Laboratory diagnosis, Treatment, diagnoses.
الانتشار. * Epidimiolgy : prevention & control.

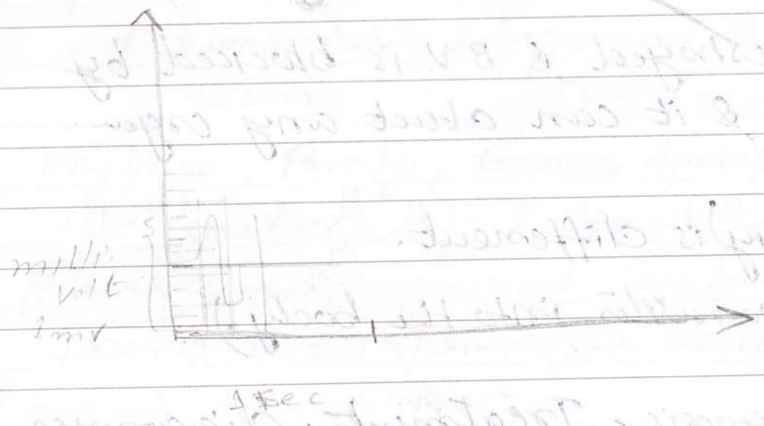
* parasitology :-

* Vaccines :- . No clear Vaccination for parasites.

1) BCE Vaccein against → Tuberculosis. } well developed
2) Measles, rubella, Yellow fever. } Vaccines.
3) TAB Vaccine → Thyphoid
Salmonela Typhy → The cause of Typhoyde.
" Partyphy A → Bacterial ~~Infecti~~ Vacinations.
" " B →
diaphteria, Tetanus, hepatytis, influenza type B, MMR

Bacterial & viral vaccines. (measles, mumps, rubella.)





(Tractus Solitarius)

End / Epi تميز ما بين

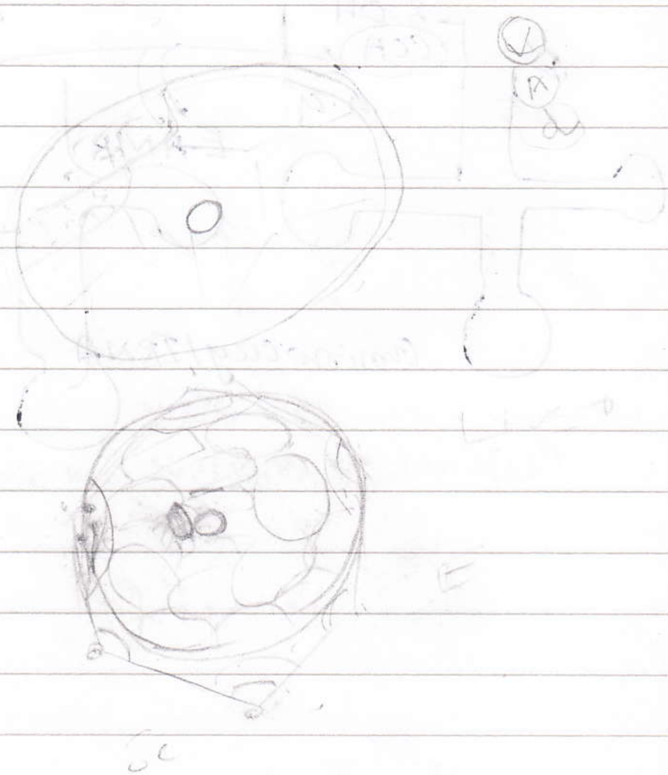
Adipose tissue.
Coronary Vessels.

Sun → Embryo. 9:10
Mon → Anat. 11:15
Tues → Histo. 9:10
Wed. → parasite. 11:15.
Thursday → physio.

24/12 → practice Histo
26/12 → Exam Histo

28/12 Sat → Bio
29 - 30 → Practic Ana.

31 → Exam Ana.



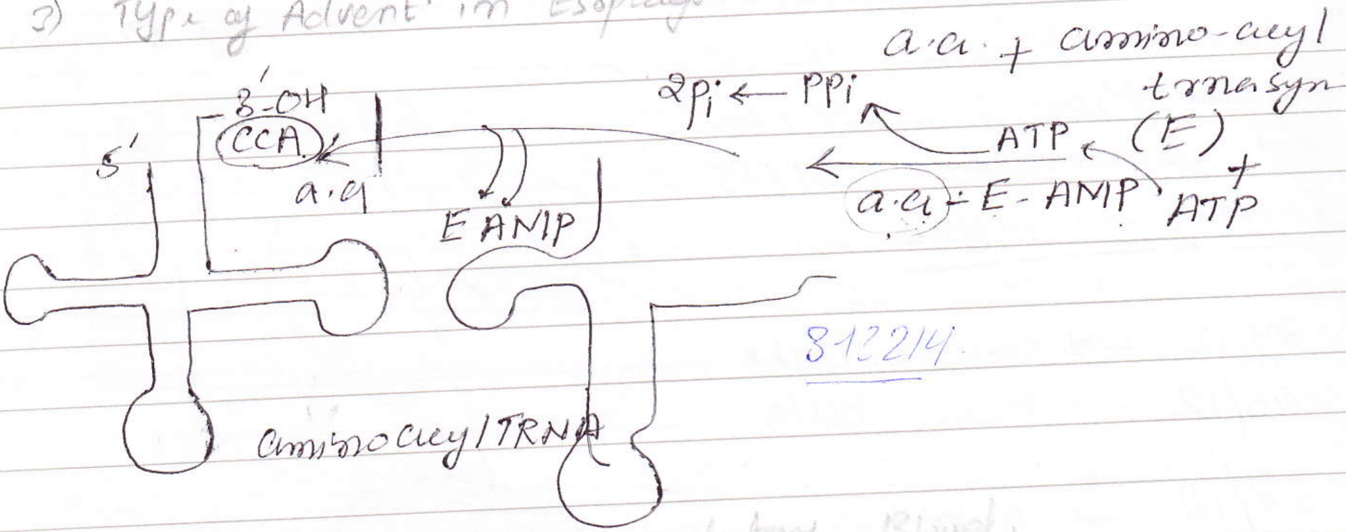
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زين العابدين المدني

1) layers of Oral cavity & is it included within GIT.

2) Type of muscle in Oral cavity.

3) Type of Advent. in Esophagus.



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Respiratory, digestive, circulatory, Blood.

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